EXAMINATION OF COGNITIVE PROCESSING THERAPY OUTCOMES IN VETERANS WITH POSTTRAUMATIC STRESS DISORDER, WITH OR WITHOUT CHRONIC PAIN, AND WITH OR WITHOUT PHARMACOLOGICAL TREATMENT FOR PAIN

BY

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Abstract

Posttraumatic stress disorder (PTSD) and chronic pain are two of the most prevalent conditions affecting the veteran population, with approximately 20% of veterans holding a PTSD diagnosis (Fisher, 2014) and roughly 55% suffering from a chronic pain condition (National Institute of Health, 2015). It is estimated that over 60% of veterans with PTSD also hold a chronic pain diagnosis (Asmundson, 2014). Scholars have theorized about the unique relationships between PTSD and chronic pain, including a mutual maintenance model (Sharp & Harvey, 2001) and a shared vulnerability model (Asmundson et al., 2002). Given this rising comorbid prevalence and associated theoretical foundation, the present study aimed to examine treatment outcomes of cognitive processing therapy (CPT), in Veterans with PTSD, with or without chronic pain, and with or without pharmacological treatment for pain, in the reduction of both PTSD and depression symptoms, utilizing the Posttraumatic Stress Disorder Checklist (PCL) and Beck Depression Inventory, Second Edition (BDI-II). A retrospective chart review resulted in a sample of 94 veterans across three distinct cohorts; Cohort 1: veterans with a chronic pain condition and pharmacological treatment for pain, Cohort 2: veterans with a chronic pain condition and no pharmacological treatment or any other identified treatment for pain, and Cohort 3: veterans without a chronic pain condition. A MANOVA was conducted using the sample's pre-treatment and post-treatment PCL and BDI-II scores to examine the symptom reduction across and among the three cohorts. Significant differences were observed between pre-treatment and post-treatment measures of both the PCL and BDI-II, across all cohorts. Significance in between-group differences on PCL score change across CPT was not significant, however demonstrated marginal significance. Between-cohort differences on BDI-II score changes were significant through the MANOVA analysis, however the post-hoc cohort mean comparison failed to reach

significance. The study discusses the significance of results in the context of existing literature as well as future directions for research.

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Chapter I

Introduction

Posttraumatic stress disorder (PTSD) is one of the most relevant topics pertaining to the Veteran population. While prevalence rates differ among certain eras of veterans, the general prevalence for PTSD in the Veteran population is approximately 20%, compared to 5% of men and 11% of women in the general population (Kessler et al., 1995). PTSD is characterized by four distinct clusters of symptoms: re-experiencing of the traumatic event, avoidance of trauma-related stimuli, emotional numbing, and hyperarousal (American Psychiatric Association, 2013). If left untreated, these symptoms become chronic and more difficult to treat (Riggs, Rothbaum, & Foa, 1995). Olatunji and colleagues (2007) describe the various negative implications PTSD can have on an individual. They state that PTSD can negatively impact an individual's physical health while also limiting their social functioning and disrupting close relationships. In the veteran population specifically, it is estimated that annual healthcare costs for veterans with PTSD are significantly higher than healthcare costs for veterans with other physical health conditions (Tuerk et al., 2012). More specifically, healthcare costs related to PTSD averaged $8,000 per veteran annually and healthcare costs related to physical conditions averaged $4,000 per veteran annually (Tuerk et al., 2012). As demonstrated, PTSD has significant impacts on both individual and societal levels. Thus, how to most effectively treat PTSD is a significant challenge facing the health care professions.

Another extremely relevant topic within the Veteran population is chronic pain. Approximately 55% of veterans suffer from some form of chronic pain, a drastic increase compared to the approximate 30% of individuals who suffer from chronic pain in the general population (National Institute of Health, 2015). Chronic pain has been defined as a complex

condition that is comprised of physiological, psychological, and social components (Casey, 2014). It is associated with both actual and perceived tissue damage and is often categorized as an unpleasant sensory-emotional experience (Otis, Pincus, & Keane, 2006). Chronic pain presents in a complex fashion in that it can manifest in a variety of distinct physical locations (i.e. back, joint, head, nerves, etc.) and, from an epidemiological perspective, can have an array of hypothesized origins (i.e. fibromyalgia, injury, muscle deterioration) (Smith, Elliott, & Hannaford, 2004). Chronic pain distinguishes itself from acute pain as it is defined as pain that persists for three or more months (International Association for the Study of Pain, 1986). Additionally, chronic pain can bring about a host of negative ramifications including vocational impairment, limited mobility, decreased quality of life, and even depression and anxiety (Ratcliffe, Enns, Belik, & Sareen, 2008; Sheu, Lussier, & Rosenblum, 2008; Turk, 2002).

Both chronic pain and PTSD have received a great deal of attention in the literature with a large portion dedicated to their presence in the veteran population. Clinical observations have shown a common phenomenon in veterans suffering from PTSD also reporting chronic pain (Asmundson et al., 2002). Research has presented information on demographic prevalence, diagnostic criteria, treatment implications, and related functional impairment for both PTSD and chronic pain. Notably, however, while a solid body of research exists on these constructs independently, a much smaller research base exists on the two as comorbid conditions. What this smaller evidence base does reveal is the inextricable link between the two conditions, specifically within the veteran population.

Link Between Chronic Pain and PTSD

While PTSD and chronic pain present challenges and repercussions as individual diagnoses, the two are also often paired as co-morbid conditions with an inextricable relationship

that presents a unique set of implications. Asmundson and colleagues (2004) discuss the complex relationship between PTSD and chronic pain as they delineate the specific associations between symptoms clusters of PTSD and facets of the pain experience. They specifically identify that the "re-experiencing" symptoms of PTSD are directly related to pain severity and the self-reporting of physical symptoms. Additionally, Asmundson and colleagues (2004) describe a negative relationship between the "numbing" symptoms of PTSD and one's ability to function amidst a low level of pain. Lastly, they illustrate the positive relationship between hyper-arousal symptoms of PTSD and detection of pain.

In addition to Asmundson and colleagues' (2004) findings, there are specific models in the literature that also delineate the relationship between PTSD and chronic pain. Sharp and Harvey (2001) introduced their mutual maintenance model to describe the complex relationship between PTSD and chronic pain. The mutual maintenance model posits that there are both physiological and psychological factors of PTSD that maintain and even exacerbate one's experience of pain while, simultaneously, there are psychological components of chronic pain that maintain and exacerbate symptoms of PTSD. Sharp and Harvey identified seven specific factors that contribute the mutual maintenance of these conditions which include factors such as attentional biases, avoidance, depression, and pain as a reminder of the trauma.

Asmundson and colleagues (2002) present a similar yet distinct model that highlights the relationship between PTSD and chronic pain. They posit that certain individual characteristics or traits predispose one to develop not only PTSD but also chronic pain after experiencing an event that is painful and traumatic. The model identifies potential vulnerability characteristics related to high anxiety sensitivity, a selective attention for threat, a higher innate sense of uncontrollability, and a lower threshold for sympathetic nervous system activation. The model

relates these characteristics by illustrating the ways in which each of these vulnerability factors makes the individual susceptible to developing both PTSD and chronic pain, not just one condition. It is also important to note that the model suggests these factors often integrate and combine which then increases the individual's susceptibility for development of both conditions.

Depression in PTSD and Chronic Pain

Depression is one of the most overtly identified co-morbid conditions for both PTSD and chronic pain. It is estimated that approximately 50% of individuals with PTSD also meet criteria for major depressive disorder (Elhai et al., 2008). One study in particular found that 88% of their sample of veterans seeking treatment for PTSD had co-morbid depression (Frueh et al., 2005). Similarly, depression has significantly high co-morbid rates with chronic pain. Roughly half (40-60%) of patients with chronic pain also meet criteria for major depressive disorder (Ohayon & Schatzberg, 2003; Kroenke et al., 2009).

In addition to the clear empirical support for the presence of these co-morbidities, the literature illustrates the specific impacts that depression has on both PTSD and chronic pain. A diagnosis of PTSD has demonstrated significant implications for the presentation and treatment of major depression. Most notably, PTSD increases the severity of depression with significantly increased risks of suicidal ideation decreased social support, and higher disability prevalence (Campbell et al., 2007). Hegel and colleagues (2005) demonstrated that, in comparison to patients with only depression, patients with both PTSD and depression had significantly delayed and less impactful responses to a primary care-based intervention for depression.

Similarly, chronic pain and depression demonstrate a significant relationship. Chronic pain has been liked to increased rates of major depression as well as increased rates of suicidal ideation and suicide attempts (Ratcliffe et al., 2008). It has also been shown that depression

symptom severity is positively associated with disability related to pain in that greater depression symptom severity is associated with greater levels of disability (Demyttenaere et al., 2006). Major depression also serves as a risk factor for developing chronic pain (Gureje et al., 2008). Zautra, Johnson, and Davis (2005) demonstrate the significance of positive affect in coping with chronic pain by building resilience, linking the experience of pain to affect and mood.

In individuals with both PTSD and chronic pain, depression can have a significant impact on level of disability and treatment progress. Sharp and Harvey (2001) allude to these implications in discussing their mutual maintenance factor of depression from their mutual maintenance model of PTSD and chronic pain. They state that depressive symptoms, specifically anhedonia and fatigue, often lead to decreased physical activity levels. Further, they describe that decreased activity levels have a two-fold impact on individuals who present with both chronic pain and PTSD; they are associated with increased pain and disability from a chronic pain perspective and they also limit the individual's exposure to trauma-related stimuli which is a critical technique in treating PTSD.

Treatment Challenges

As demonstrated, PTSD and chronic pain as comorbid conditions appear to have a perpetual relationship that affects symptom severity as well as treatment effectiveness. Although the literature supports and even highlights this unique relationship, current treatment regimens fail to conceptualize a PTSD with chronic pain presentation from a dual-diagnosis perspective and therefore fail to adequately address the unique components of their comorbidity. As these conditions largely stem from different professional fields, care is often left uncoordinated. Mental health professionals do not routinely screen for physiological conditions such as chronic pain and healthcare professionals often do not screen for mental health conditions such as PTSD.

The lack of professional coordination leaves this comorbidity largely unaddressed in current treatment protocols (Asmundson, 2014). A study done by Farmer and colleagues (2009) examined the treatment effects of a collaborative care approach to treating chronic pain and co-occurring anxiety disorders (generalized anxiety disorder and panic disorder) in comparison to treatment as usual in a primary care clinic. Their initial investigation found that when an individual's pain was severe enough to interfere in daily functioning, treatment effectiveness for the individual's co-occurring anxiety disorder significantly decreased.

In their mutual maintenance model of PTSD and chronic pain, Sharp and Harvey (2001) identify the way in which both conditions independently command a significant amount of cognitive demand and, when coupled together, they utilize a majority of an individual's attentional capacity. It is reasonable to expect that individuals who present with both PTSD and chronic pain will demonstrate a decreased ability to capitalize on the cognitive strategies presented in the empirically based treatments for PTSD, due to the high level of attentional demand that chronic pain also requires. These approaches ask individuals to identify their maladaptive thought processes and challenge automatic thought patterns. While this has been shown to be effective in treating these conditions separately, the increased cognitive demand from the comorbid conditions reduces individuals' available cognitive capacity, making it more difficult for them to engage in cognitive strategies and rendering the treatment less likely to be effective (Sharp & Harvey).

Conversely, Sharp and Harvey (2001) demonstrate the way in which PTSD can inhibit treatment of chronic pain. Their mutual maintenance model of PTSD and chronic pain identifies that increased anxiety can exacerbate pain. Here, Sharp and Harvey allude to the psychological component of pain when discussing the impact of anxiety on one's pain perception. They argue

that simply the experience of anxiety can exacerbate one's experience of pain. As PTSD is characterized by high levels of anxiety, the simple nature of its comorbidity with chronic pain is expected to heighten the individual's experience of pain.

There is no one specific treatment that has been validated to treat both PTSD and chronic pain in conjuction. However, based on the interplay of the symptoms between PTSD and chronic pain, it can be reasonably argued that treating one condition would have implications for the other, especially in treating the overlapping symptoms between the two. Further, it can also be argued that treating both conditions simultaneously would maximize the treatment effect for those who experience both conditions. This study aims to examine group differences in treatment outcomes following cognitive processing therapy (CPT) between veterans with PTSD, with or without chronic pain, and with or without pharmacological treatment for pain.

Cognitive processing therapy (CPT) is a 12-session manualized psychological intervention that addresses maladaptive cognitive appraisals of the traumatic event. It has been distinguished as an efficacious treatment for PTSD, and more specifically, within the Veteran population (Suris, Link-Malcolm, Chard, Ahn, & North, 2013; Resick, Monson, & Chard, 2007). Cognitive processing therapy (CPT) also shares distinct similarities with cognitive behavioral therapy for chronic pain (CBT-CP), an evidenced-based psychological intervention for chronic pain. Both approaches utilize cognitive strategies such as exploring and identifying automatic appraisals of either the trauma or experiences related to pain. In both approaches, individuals are encouraged to explore the way in which their cognitive appraisals are impacting their emotional experiences related to the specific focus of treatment (trauma or pain). Because of its empirical support and wide implementation in Veterans Affairs Medical Centers, as well as its similarities to cognitive-behavioral therapy for chronic pain, CPT was selected as the primary treatment

regimen of interest for this study. Additionally, the other component to the dual-disciplinary approach will be pharmacological treatment for pain, which will be used in conjunction with CPT.

Statement of the Problem

As previously discussed, it is evident that PTSD and chronic pain share an inextricable link. Several models in the literature highlight the link between PTSD and chronic pain and further illustrate the implications each has for the other. However, literature is lacking in terms of effective treatment for both unique and shared symptoms of PTSD and chronic pain when both present co-morbidly. Intuitively, it seems reasonable to expect that when both conditions are treated simultaneously, the treatment outcome for reducing symptoms of either condition would be greater than when the other condition is untreated. However, the study also acknowledges the potential that significant reduction in symptoms are seen across all cohorts in the study, some with single treatment and some with simultaneous treatments. The current study will empirically examine this phenomenon, hoping to produce evidence that warrants clinical and empirical attention to the unique aspects and treatment considerations for this comorbidity.

The Present Study

This study aimed to examine the reduction of both unique symptoms of PTSD and the overlapping symptoms of PTSD and chronic pain, identified through assessments of both PTSD and depressive symptoms, following engagement in CPT for PTSD, with or without chronic pain, and with or without pharmacological treatment for pain. While it would have been beneficial to also explore PTSD and depression symptom reduction following engagement in pharmacological treatment of pain without CPT, data is limited in this area, as desired outcome measures are not assessed in routine pharmacological treatment of pain. A retrospective analysis

was conducted utilizing an existing database from the Veterans Affairs Informatics and Computing Infrastructure (VINCI). The study will examine the differences in PTSD symptom change and depressive symptom change over the course of CPT between veterans with PTSD and a chronic pain condition treated with pharmacotherapy, and veterans with PTSD and a chronic pain condition without pharmacological treatment for pain, veterans with PTSD and no chronic pain condition. The specific research questions to be addressed in the present study include the following:

Research Questions and Hypotheses

Research Question I: Are there significant differences between pre- and post-CPT measurements of PTSD symptom severity?

Hypothesis I: There are significant differences between pre- and post-CPT measurements of PTSD symptom severity, regardless of veterans' chronic pain status.

Research Question II: Are there significant differences between pre- and post-CPT measurements of depression symptom severity?

Hypothesis II: There are significant differences between pre- and post-CPT measurements of depression symptom severity, regardless of veterans' chronic pain status.

Research Question III: Are there significant differences in the degree of PTSD symptom change, between three cohorts of veterans: 1) veterans with a chronic pain condition and pharmacological treatment for pain, 2) veterans with a chronic pain condition without pharmacological treatment for pain, and 3) veterans with no chronic pain condition and no pharmacological treatment for pain, following engagement in CPT?

Hypothesis III: There are significant differences in the degree of PTSD symptom change between the three cohorts of veterans: 1) veterans with a chronic pain condition and

pharmacological treatment for pain, 2) veterans with a chronic pain condition without pharmacological treatment for pain, and 3) veterans with no chronic pain condition and no pharmacological treatment for pain, following engagement in CPT, with Cohort 2 demonstrating significantly less PTSD symptom change than Cohorts 1 and 3.

Research Question IV: Are there significant differences in depression symptom change, between the three cohorts of veterans: 1) veterans with a chronic pain condition and pharmacological treatment for pain, 2) veterans with a chronic pain condition without pharmacological treatment for pain, and 3) veterans with no chronic pain condition and no pharmacological treatment for pain, following engagement in CPT?

Hypothesis IV: There are significant differences in depression symptom change between the three cohorts of veterans: 1) veterans with a chronic pain condition and pharmacological treatment for pain, 2) veterans with a chronic pain condition without pharmacological treatment for pain, and 3) veterans with no chronic pain condition and no pharmacological treatment for pain, following engagement in CPT, with Cohort 2 demonstrating significantly less depressive symptom reduction than Cohorts 1 and 3.

Chapter II

Review of the Literature

The current chapter serves to provide information related to the theoretical and empirical background of both posttraumatic stress disorder (PTSD) and chronic pain as well as information related specifically to the relationship between PTSD and chronic pain. Unique information related to each condition includes diagnostic criteria, prevalence, and associated negative implications. Information pertaining to the relationship between PTSD and chronic pain is presented following each unique condition's material and it discusses specific theories that aim to describe the distinct relationship the two share. Moreover, the literature on existing treatments and associated efficacy for these conditions is included. Lastly, a brief summary is provided detailing the primary findings and limitations of the literature.

Posttraumatic Stress Disorder (PTSD)

Definition and diagnostic criteria. The Diagnostic and Statistic Manual of Mental Disorders, 5th Edition, defines PTSD as a psychological condition that develops following exposure to actual or threatened death, serious injury, or sexual violence and is characterized by four distinct clusters of symptoms (American Psychiatric Association, 2013). Individuals who have had exposure to trauma must exhibit symptoms of re-experiencing the traumatic event, which likely involves flashbacks and nightmares of the traumatic event. Additionally, individuals display marked attempts to avoid stimuli related to the traumatic event. Individuals with PTSD will also experience negative alterations in cognition or mood following the traumatic event. Lastly, individuals with PTSD will develop significantly heightened arousal and reactivity. At least one symptom of both re-experiencing and avoidance as well as at least two symptoms of negative alteration in mood or cognition and arousal must be present for at least one month

following the trauma and the symptoms must cause significant functional impairment to meet full criteria for the PTSD diagnosis (American Psychiatric Association, 2013).

The lifetime prevalence for PTSD in the general population is approximately 8% (American Psychiatric Association, 2013), however, for veterans and military personnel, this rate is much higher. Most uniformly, it is estimated that between 15% to 25% of veterans and military personnel will develop PTSD at some point in their lifetime (Center for Disease Control, 1988; Hoge et al., 2004, Ramchand et al., 2010). While these rates seem consistent across various findings, Fisher (2014) illustrates the controversy surrounding the accurate identification of PTSD prevalence in the Veteran population. He notes that given the culture of the military, soldiers and veterans are often hesitant or resistant to disclosing their mental health concerns due to perceived or actual stigma. Furthermore, soldiers and even veterans are often hesitant to disclose, as they fear a disclosure of their mental health condition will prohibit them from returning to duty and ultimately result in a discharge from the military. Per this fear, Fisher argues that PTSD often goes unreported and undetected in a significant group of soldiers and veterans, suggesting that our current prevalence rates are a considerable underestimation.

Consequences of posttraumatic stress disorder. Due to its prevalence, particularly within the veteran population, PTSD poses a significant healthcare burden in the United States. In a recent study done by Tuerk and colleagues (2012), it was found that PTSD in veterans presents a greater number of missed days from work, higher public health costs, and greater healthcare utilization across disciplines than physical health conditions in veterans. More specifically, Tuerk and colleagues identified that healthcare costs related to PTSD were approximately $8,000 per veteran annually, while healthcare costs related to physical health conditions were, on average, $4,000 per veteran annually. Deykin and colleagues (2001) support

this notion with their finding that PTSD is associated with significantly higher rates of service use as well as medical and social costs.

In addition to the implications PTSD poses for the healthcare system at large, PTSD brings about a variety of negative consequences for the individuals who have it. PTSD has been associated with a variety of different mental health conditions as well as a greater level of overall distress for individuals. In their sample of Vietnam veterans seeking treatment for PTSD, Frueh and colleagues (2005) found that 88% of their sample also met criteria for major depression, 42% had a co-morbid substance use disorder, 7% had some form of psychosis, and 15% had an additional anxiety disorder separate from PTSD. Additionally, Shea, Vujanovic, Mansfield, Sevin, and Liu (2010) found that symptoms of hyperarousal in PTSD served as significant predictor of subjective distress. They illustrate how hyperarousal can lead to difficulty concentrating, intense feelings of anger, and difficulty with sustained sleep leading to increased fatigue. Each of these facets contribute to individuals' overall well-being leaving room for significant distress when impaired.

PTSD has also demonstrated a significant negative impact on individuals' physical health status. David and colleagues (2004) found, in their examination of male veterans with PTSD and male veterans with alcohol dependence, that those with PTSD had higher rates of osteoarthritis, diabetes, cardiac disease, obesity, and higher cholesterol than those with alcohol dependence. Additionally, Kimerling and colleagues (2000) found that female veterans with symptoms of hyperarousal had correlated negative perceptions about their physical health, which has been shown to lead to poorer health outcomes. It has also been demonstrated that individuals with PTSD who are seen in primary care and non-psychiatric settings report more severe medical

symptoms than individuals who do not have PTSD (Hankin et al., 1996). It is clear that PTSD has direct implications for medical care and physical health status.

PTSD can also cause impairment in social and intimate relationships. This particular relationship between PTSD and relationship impairment is well documented in the literature and has been demonstrated in combat veterans across all war eras (Monson et al., 2009). Veterans with PTSD have higher divorce rates than trauma-exposed veterans without PTSD (Cook et al., 2004) and are more likely to demonstrate verbal and physical aggression against their partners (Sherman et al., 2006). Much of the research in this area illustrates the role of the emotional detachment and numbing symptoms of PTSD in relationship distress. Shea and colleagues (2010) describe how these symptoms are often responsible for individuals' inability to fully experience emotions ultimately decreasing their empathetic capabilities. Shea and colleagues also note that numbing and emotional detachment decrease individuals' willingness to disclose emotions and be open with their partners. This lack of openness and vulnerability significantly limits the capacity of the relationship. It has also been demonstrated that veterans with PTSD exhibit greater anxiety related to intimacy than veterans without PTSD (Riggs et al., 1998). As conflict arises, particularly within partner relationships, individuals receive reduced amount of support further perpetuating their symptoms of avoidance and numbing.

Additionally, PTSD has demonstrated significant negative impacts on vocational and daily functioning. As previously discussed, hyperarousal symptoms of PTSD produce greater levels of overall distress in individuals and cause impairment in sleep, concentration, and emotion regulation which often result in decreased productivity and occupational impairment (Shea et al., 2010). Martz and Cook (2001) also provide insight into the impairments that PTSD can pose on occupational functioning. They illustrate the way in which certain vocational tasks

can trigger memories related to the traumatic event. For example, Martz and Cook identify that working with heavy machinery may remind a veteran with PTSD of military equipment and trigger traumatic memories related to their service. Additionally, working in medical setting and seeing injured individuals could serve as a trigger for individuals with PTSD. PTSD is associated with a host of negative implications for vocational functioning in addition to other aspects of wellbeing.

Theories of posttraumatic stress disorder. Per its prevalence and distinct clinical presentation, PTSD has warranted a great deal of attention in psychological literature. As such, a plethora of theories have been established to conceptualize the etiology and manifestation of PTSD. One of the foundational theories of PTSD is Foa and Kozak's (1986) emotional processing theory. Emotional processing theory illustrates the role that complex fear structures in memory play in the development of PTSD. Foa and Kozak describe how fear is uniquely depicted in memory as a network that incorporates information regarding what stimuli or situations should be feared, appropriate physiological and behavioral responses to fear, and the meaning of the feared stimuli or responses. This information is theorized to provide the individual with the capacity to identify the need for escape or avoidance behavior. Foa and Kozak acknowledge that fear structures in memory serve an adaptive purpose as, in healthy individuals, they allow for the appropriate detection of danger and advantageous avoidance behaviors. However, they further describe the pathological properties of these fear structures that manifest with PTSD. In individuals with PTSD, these fear structures begin to overgeneralize stimuli as fearful and as a result, individuals respond to benign stimuli with a fear response. Foa and Kozak link these maladaptive processes to individuals' negative beliefs that surround aspects of the trauma, noting that most often individuals begin to believe that the world is a highly

dangerous place. Furthermore, Foa and Kozak describe the way in which emotional processing allows individuals to modify this belief and restore adaptive, non-generalized fear structures, ultimately reducing fear and fear responses. They state that individuals must first be presented with fear-relevant information to activate the fear structure and allow it to be modified. Following this activation, individuals must be presented with new information that is incompatible with the existing fear structure as this will force individuals to modify the evoked fear structure, ultimately restoring congruency in the fear structure and reducing fear responses. Foa and Kozak's arguments support the use of exposure techniques in treating anxiety disorders.

Brewin and colleagues (1996) offer a distinct theory of PTSD labeled dual representation theory. Brewin and colleagues suggest PTSD is maintained and represented by two different types of memories; verbally accessible memories and situationally accessible memories. Verbally accessible memories are defined as memories that can be deliberately retrieved and verbally recalled to describe specific recollections, emotional experiences, and perceived meaning related to the trauma. Conversely, situationally accessible memories cannot be deliberately accessed and remain unconscious until triggered by stimuli related to the traumatic experience. Situationally accessible memories are most often associated with flashbacks of the traumatic event and are characterized as unsolicited and even intrusive. Brewin and colleagues argue that while these types of memories are distinct, an individual has the capacity to experience them simultaneously in addition to individually. Similar to Foa and Kozak's emotional processing theory, Brewin and colleagues argue that successful emotional processing is the key to fear reduction but add that this process is contingent upon exposure to situationally accessible memories. They illustrate the role that situationally accessible memories play in accurately identifying sensory information and adjusting beliefs about the traumatic experience.

Additionally, Ehlers and Clarke (2000) offer a cognitive model of PTSD. In general terms, the cognitive model of PTSD postulates that particularly negative appraisals of the traumatic event foster the development of PTSD. Ehlers and Clark argue that individuals with PTSD are unable to view their trauma as a time-limited past event that does not have grave implications for their future. Ehlers and Clark describe the way in which these negative appraisals develop by stating that individuals can develop both internal and external negative appraisals that impact both how they view themselves and how they view the world. Most commonly, individuals with PTSD develop negative external beliefs about future events and the general state of the future in which they begin to view benign situations as harmful and overestimate the probability of future traumatic or disastrous events. Additionally, individuals with PTSD develop negative internal appraisals as a result of the trauma that place blame on themselves for the trauma occurring and often introduce serious doubts about their ability to remain safe. Both of these types internal and external types of appraisals instill a sense of ongoing threat for individuals with PTSD, bringing about symptoms associated with fear and prolonged arousal. The aim of the cognitive approach to treating PTSD is to specifically identify these negative, maladaptive appraisals and introduce new information that negates these beliefs.

Ehlers and Clark briefly discuss the behavioral consequences associated with these negative appraisals. Most notably, they identify the increased "safety behaviors" that these individuals often engage in as they operate under the assumption or appraisal that future danger is highly likely. This can include keeping a weapon close to their bed, excessively checking locks, staying up during the night to "keep watch", and many others. Additionally, they illustrate that, in an attempt to reduce emotional distress, individuals with PTSD often engage in both

physical and mental avoidance strategies to avoid any potentially threatening situation as well as the experience of any negative emotional experiences.

Treatment of posttraumatic stress disorder. PTSD has been demonstrated as a severe and persistent condition with considerable capability to cause death via suicide as well as serious impairments in psychosocial functioning (Johnson et al., 2004). As such, treatment of PTSD becomes an important area of research. Monson and colleagues (2006) state that even when chronic, PTSD is treatable across all demographics. On a similar note, Creamer and Forbes (2004) identify the potential for treatment of chronic PTSD while also stating that PTSD, if left untreated, has the potential to progress to the point at which pre-trauma functioning is highly unlikely.

There is an array of treatment options available for PTSD that span a variety of different disciplines. The literature has examined and occasionally demonstrated some preliminary efficacy for alternative treatments of chronic pain including medication (Stein et al., 2006), other psychological interventions such as mindfulness (Banks et al., 2015), and even trauma-focused yoga (Mitchell et al., 2014). While there are a host of different treatment options available for PTSD, only a select few have demonstrated considerable empirical support for their effectiveness.

One of the evidence-based approaches for PTSD is cognitive processing therapy (CPT). While it was originally created to treat rape-related PTSD, CPT has been specifically adapted to treat PTSD in the veteran population (Resick, Monson, & Chard, 2007) and has demonstrated efficacy with a variety of demographic groups within the veteran population (Foa et al., 2008). As an example of this efficacy, Monson and colleagues (2006) found in their sample of combat veterans that over half of the sample showed significant decreases in PTSD symptoms and 40%

no longer met full diagnostic criteria for PTSD following a regimen of CPT. This, along with similar findings, has established CPT as a best-practice model identified by the International Society for Traumatic Stress Studies (Foa et al., 2008).

The aim of CPT is to expose individuals to their traumatic memories via writing about and verbally recalling the trauma with a specific emphasis on the associated cognitions and emotions. The basic components of the treatment can be described in three distinct phases. The first phase of cognitive processing therapy aims to explore the impact of the trauma and provides the therapist an opportunity to educate the client about the relationship between cognitions and emotions. Phase two of CPT involves written summaries of the trauma in which the individual and the therapist examine the specific appraisals the individual has made of the traumatic event. Here, the therapist works to help the individual identify where he or she may be "stuck" in terms of his or her cognitive appraisals of the trauma. A very common example of a "stuck point" is when the individual assumes an overt amount of self-blame for all or certain aspects of the traumatic event and develops appraisals such as, "I could have prevented this." The final stage of CPT is comprised of a more in-depth analysis of the cognitive distortions in the individual's appraisal of the trauma and remediation for those distortions.

Chronic Pain

Chronic pain is one of the most prevalent health conditions in the United States with more than 100 million Americans currently suffering from some form of chronic pain (Institute of Medicine, 2011). As such, chronic pain is responsible for a substantial amount of healthcare costs and days of work missed annually. It is estimated that chronic pain conditions cost the United States an estimated $600 billion dollars in lost productive time and healthcare costs each year (Toblin et al., 2010). In the Veteran population specifically, it is estimated that 55%

experience some form of chronic pain, compared to 30% of the general population (National Institute of Health, 2015). Chronic pain's elevated presence in the veteran population also leads to significant costs associated with healthcare and disability compensation for both chronic pain and its associated conditions (i.e. depression) (Toblin et al.).

Definitions and diagnostic criteria. The International Association for the Study of Pain (1986) identifies pain as "an unpleasant sensory and emotional experience that is associated with actual or potential tissue damage." Chronic pain is loosely defined as pain that persists beyond three to six months and is likely to increase in pathophysiology complexity over time (International Association for the Study of Pain). As chronic pain offers a loose and widely applicable definition, there are several diagnostic labels that meet criteria for chronic pain. Diagnostic labels range from location-specific (i.e. chronic low back pain) to general pain conditions (i.e. other chronic pain, chronic pain syndrome) but generally include a vague and minimal set of criteria that are largely contingent upon the individual's reported pain levels.

Historically, pain has been evolutionarily favorable in that it motivates individuals to avoid potentially harmful or threatening scenarios and engage in recovery behaviors as needed. Nagasko, Oaklander, and Dworkin (2003) describe the importance of pain in discussing congenital analgesia, a rare genetic disease that is characterized by the inability to experience pain. They describe the significant risk associated with this disease, as it does not allow individuals the capacity to recognize the severity of injuries that warrant immediate attention and treatment. Nagasko, Oaklander and Dworkin note that individuals with congenital analgesia do not often survive past childhood as they develop serious injuries that go undetected and therefore untreated.

While acute pain has adaptive properties, pain that becomes chronic loses these adaptive properties and causes considerable distress and impairment. In responding to chronic pain as if it were acute, individuals remain increasingly focused on the pain as they aim to identify its origin and etiology in an effort to develop a plan for treatment. As chronic pain is often complex and incurable, the individual is left indefinitely searching for this information, consuming cognitive, emotional, and physical resources. Additionally, individuals with chronic pain adapt prolonged avoidance strategies, which, over time, develop into associated fears of a larger array of potentially harmful activities, limiting their overall activity levels and significantly impairing their social functioning (Gureje, Von Korff, Simon, & Gator, 1998).

Consequences of chronic pain. Chronic pain has been shown to impact a variety of different facets of an individual's wellbeing. One of the most prominent findings in this area is the negative impact of chronic pain on mood. Several studies illustrate the direct correlation between chronic pain and major depressive disorder (Ratcliffe et al., 2008; Banks & Kerns, 1996; Zautra, Smith, Affleck, & Tennen, 2001). Additionally, chronic pain has been strongly associated with increased rates of suicidal ideation and suicide attempts (Ratcliffe et al., 2008). To supplement these findings, Finucane and colleagues (2012) found in their analysis of basic emotion profiles of healthy individuals, individuals with major depressive disorder, individuals with chronic pain, and individuals with PTSD that chronic pain patients and depressed patients shared remarkably similar emotion profiles. They note that the individuals with chronic pain and the individuals with major depressive disorder only differed in the significant presence of anger, which was found predominantly in the chronic pain sample.

There is evidence that chronic pain also affects cognitive processes and brain function. Grachev, Fredrickson, and Apkarian (2002) explored these implications and found that chronic

pain was associated with several neurological distinctions. They found that individuals with chronic pain have less activation in their default mode networks, abnormal brain chemistry, and a loss of neocortical gray matter. Grachev, Fredrickson, and Apkarian argue that each of these distinctions are related to the way in which chronic pain is encoded in the brain as well as the subsequent behavioral responses. In other words, these neurological alterations can change an individual's pain experience, likely through his or her perception of the pain and how it globally impacts him or her as an individual. This can take form through interpretation of pain severity, conceptualizations of disability, and anticipations of recovery. This is of particular importance when noting that pain-related thoughts and beliefs are associated with psychological functioning (Stroud et al., 2000), physical functioning (Turner et al., 2000), coping efforts (Williams et al., 1996), and response to treatment (Tota-Faucette et al., 1993).

Sleep is also significantly impacted by chronic pain. Marin, Chyan, & Miklos (2006) found that, in a sample of chronic low back pain patients, pain intensity was directly correlated to sleep disturbance. Tang and colleagues (2007) found that approximately 75% of individuals with chronic pain also suffered from difficulties with sleep. Physiologically, the experience of pain itself presents a significant barrier to sleep in that pain signals demand cognitive attention, inhibiting a sleep state (Ohayan, 2005). Individuals with chronic pain are more likely to experience insomnia, even when controlling for depression, anxiety, and other medical conditions (Taylor et al., 2007).

Additionally, there is some evidence to suggest that chronic pain affects cardiovascular health. Bruehl and colleagues (2005) found that intensity of chronic pain served as a statistically significant predictor for hypertension even after controlling for demographic variables such as age, gender, race, and family history. To supplement this finding, Chung and colleagues (2008)

identified that the baroreflex, a homeostatic, biological mechanism that is responsible for regulating blood pressure, is involved in the physiological suppression of pain. In healthy individuals, the baroreflex contributes to hypoalgesia, decreasing an individual's sensitivity to painful stimuli. In individuals with chronic pain, the baroreflex is often impaired leading to hypertension and an increased sensitivity to pain (Chung et al.).

It is also often noted in the literature that chronic pain can impair an individual's sexual function. Ambler and colleagues (2001) note that chronic pain is attributed to a variety of different concerns that impair sexual function. They describe that sexual arousal itself can be difficult with a chronic pain condition due to consistent and competing pain signals in the brain. Additionally, individuals with chronic pain may have difficulty with confidence, establishing a comfortable position, and a fear of worsening their pain, each of which can contribute to sexual dysfunction. It is important to note that sexual dysfunction, especially when sustained can cause distress within the intimate relationship (McCarberg et al., 2008).

Psychological theories of chronic pain. As previously illustrated, chronic pain has a host of negative implications, including those related to mental health. While chronic pain is often considered a biological condition, the relationship between chronic pain and mental health is well documented in the literature and several psychological theories of chronic pain have been established.

One of the most prominent psychological theories of chronic pain draws upon principles from the operant theory of conditioning. Fordyce (1996) describes the basic premise of this theory in stating that pain behaviors, such as vocalizing distress, withdrawing from activities, taking pain medication, and pain related body postures, are influenced by learning and experience, just as any behavior. Sanders (1996) further illustrates this concept in identifying a

number of different mechanisms that provide both positive and negative reinforcement for the maintenance of pain behaviors. Sanders describes the positive reinforcement that comes from increased affection and attention from caregivers when one experiences pain. Moreover, he illustrates how missing work and being relieved of normal daily responsibilities serve as negative reinforcement for pain behaviors, which perpetuates their presence.

Additionally, the experience of pain and subsequent pain behaviors can be conceptualized utilizing cognitive-behavioral theory. The cognitive-behavioral approach to pain argues that the amount of debilitation and distress the individual endures related to their pain is contingent on the way the individual interprets the pain experience and the way in which he or she copes or reacts to it (Keefe et al., 1989). Moreover, Sharp and Harvey (2001) state that chronic pain is truly a biopsychosocial phenomenon in which part of its presentation is due to the meaning the individual ascribes to the somatic aspects of his or her pain. McCracken and colleagues (1998) support this notion by stating that the extent to which the individual becomes distressed by his or her pain is a function of they way in which he or she responds to the pain and the associated attention it receives. As an example, they argue that the more distressing an individual views his or her pain to be, then the more attention he or she places on the pain and the more he or she avoids any potentially painful stimuli, ultimately increasing his or her level of disability. The cognitive-behavioral theory specifically addresses the individual's appraisal of the pain and argues that this appraisal determines the individual's behavioral response, which is often indicative of the individual's ability to cope with their pain.

Treatment of chronic pain. As previously discussed, chronic pain is a complex and often persistent condition that affects multiple areas of an individual's life. As such, the effective treatment of chronic pain proves to be incredibly important yet quite difficult. There are several

identified approaches to treating chronic pain conditions. Casey (2014) identifies the wide array of approaches available to treating chronic pain, which, aside from pharmacological treatment, includes physical therapy, acupuncture, chiropractic interventions, biofeedback, and psychological interventions. While there are a host of different treatment options, by far the most prevalent approach to treating chronic pain is pharmacological (Manchikanti et al., 2010).

Because of the complexity of chronic pain, there is little evidence of efficacy for use of a particular drug to treat chronic pain. Additionally, due to the wide array of chronic pain conditions, it is difficult to conduct strong clinical trials (Casey, 2014). However, even without specific drug-condition partnerships, the World Health Organization (1986) offers providers an "analgesic ladder" as a guide for treating chronic pain pharmacologically. The "analgesic ladder" illustrates the recommended step-wise progression that begins with considering either a paracetamol (acetaminophen) or a nonsteriodal anti-inflammatory drug (ibuprofen, naproxen, or asprin). The next step is to consider the use of both an paracetamol and a nonsteriodal anti-inflammatory drug, which, if unsuccessful, leads to the introduction of a mild opioid medication. If the mild opioid proves unsuccessful, a stronger opioid medication is introduced. The World Health Organization (1986) notes that at any point, non-conventional drugs can be introduced as a means to increase the efficacy of the analgesic.

Several different classes of medication have demonstrated efficacy in chronic pain populations. One of the most prevalent classes of medication for pharmacological treatment of chronic pain is opioid medications. Several studies have demonstrated the efficacy of opioid medication in treating chronic pain (Manchikanti et al., 2010; Vissers et al., 2010). Opioid treatment usually begins with lower-efficacy medications and as pain persists develops into use of higher-efficacy opioids (Vissers et al., 2010). Nonetheless, opioid medications demonstrate

efficacy for a variety of different types of chronic pain presenting in a vast array of severity (Manchikanti et al.). Additionally, there has been some evidence supporting the efficacy of both antidepressants and anticonvulsants in treating chronic pain (Dworkin et al., 2007). These classes of medications would fall into the World Health Organization's (1986) "non-conventional" class of medication in pain treatment, establishing that they can be introduced at any point during the pharmacological treatment regimen for chronic pain. Mease and colleagues (2010) demonstrate that specifically, antidepressants belonging to the selective norepinephrine reuptake inhibitor and tricyclic classes have the greatest amount of efficacy in treating chronic pain, compared to other antidepressants and anticonvulsants. While both opioid and antidepressant medications are beneficial in treating symptoms of pain, they have no demonstrated treatment effects for PTSD. Therefore, it is likely that individuals with both chronic pain and PTSD will require a more comprehensive treatment approach to improve symptoms of PTSD.

Chronic Pain and Posttraumatic Stress Disorder

While it's clear that both chronic pain and PTSD present unique challenges as individual conditions, there is a distinct set of challenges that arise when these conditions present comorbidly in an individual. The literature reports that between 40 to 80% of veterans with PTSD also suffer from some form of chronic pain (Morasco et al. 2013; Beckham et al., 1997), in comparison to 15-25% of individuals in the general population. Giesser and colleagues (1996) identified that, in relation to patients with only chronic pain, patients with both PTSD and chronic pain reported significantly higher pain severities and greater disability due to pain. PTSD has been found to have one of the strongest relationships with somatization (Andreski et al., 1998), which leaves room for the exacerbation of the condition in the presence of chronic pain. Asnaani and colleagues (2014) note that, because PTSD is responsible for more loss of

productivity and greater healthcare costs than other physical conditions, research should be increasingly devoted to exploring the way in which mental health symptoms impact physical health functioning. Further, in their analysis of the effect of PTSD symptoms on health-related quality of life in soliders returning from deployment (to Iraq and Afghanistan), Asnaani and colleagues found that PTSD symptoms of re-experiencing were strongly associated to individuals' physical health functioning and experience of pain. While the literature leaves room for growth in this area, there are a few theories that illustrate the unique relationship between these two conditions and the distinct implications their dual-diagnosis presents.

Mutual maintenance model. Sharp and Harvey (2001) illustrate the distinct relationship between PTSD and chronic pain in their mutual maintenance model. The model is comprised of seven mutual maintenance factors that perpetuate both PTSD and chronic pain. First, Sharp and Harvey (2001) postulate that individuals with both chronic pain and PTSD tend to develop attentional biases towards opportunities for danger or harm as well as opportunities for pain. These attentional biases perpetuate symptoms of hypervigilence ultimately leading to muscle tension, increased anxiety, and a decreased quality of life (Difede et al., 1997). Sharp and Harvey note that individuals with PTSD and chronic pain experience this attentional bias twice as often as individuals with only one condition, which ultimately exacerbates their hypervigilence and the associated negative consequences (i.e. muscle tension, increased anxiety, and decreased quality of life).

Next, Sharp and Harvey identify increased anxiety sensitivity as a perpetual factor for both conditions. They describe anxiety sensitivity as "the tendency to interpret symptoms of anxiety as indicative of harm." Sharp and Harvey argue that in PTSD and chronic pain, anxiety sensitivity fuels the misinterpretation of physical sensations leading to assumptions of

catastrophe and threat. Anxiety sensitivity has also been identified in other areas of the literature as a core predispositional factor for the development of both PTSD and distress related to chronic pain showing its strong role in the co-occurrence of these conditions. Again, as there are noted physical sensations present for both PTSD (i.e. arousal symptoms such as heart racing, muscle tension, dizziness, nausea, etc.) and chronic pain (i.e. muscle tensions or spasms, throbbing pain, headaches, etc.), anxiety can have a two-fold impact on the individual, which often further perpetuates the conditions and associated maladaptive responses.

One of the most critical components of perpetuation in this model is labeled "pain as a reminder of the trauma." Sharp and Harvey describe how sensations of pain, especially when directly linked to a traumatic injury, can often trigger memories of the trauma itself and increase the severity of PTSD symptoms. When these symptoms become more severe, they then perpetuate other aspects of the model including attentional biases or hypervigilence. To illustrate, Sharp and Harvey describe how often a pain sensation will serve as a trigger or reminder of the trauma, which often leads to avoidance of the attributed cause of that pain sensation as well as any reminders of that particular traumatic memory. As avoidance behaviors increase, the anxiety regarding these sensations increases and ultimately the conditions are perpetuated, worsening the individual's psychological health and overall impairment.

Avoidance is another crucial component of the mutual maintenance model. Sharp and Harvey illustrate how avoidance mechanisms are two-fold with this dual diagnosis. As previously described, individuals with both PTSD and chronic pain often avoid any activities or circumstances that may cause more physical pain but they also avoid any situation that may be threatening or pose danger. The maladaptive role of avoidance in these conditions is increased when the conditions present co-morbidly, as are the negative implications. As a result of the

increased avoidance behaviors, individuals with this dual diagnosis experience greater impairment in vocational, social, and interpersonal functioning, and ultimately increased their fear of pain or trauma related stimuli (Sharp & Harvey, 2001).

Next, Sharp and Harvey identify depression as a contributing factor to the perpetual relationship between PTSD and chronic pain. Depression is a common co-morbid condition to both chronic pain and PTSD. The depressive symptoms of fatigue and anhedonia often result in a lack of activity, which, for chronic pain patients, is often associated with increased pain and disability and, for PTSD patients, inhibits their exposure to trauma-related stimuli, which is a critical component to treating PTSD. Several other studies support this component of Sharp and Harvey's model by illustrating the link between both chronic pain and PTSD. Chronic pain has been linked to increasing rates of major depression and suicide attempts (Ratcliffe et al., 2008). It has also been demonstrated that chronic pain presentations often mimic depressive symptoms with anger as one of the only distinct emotional components (Finucane et al., 2012). Similarly, PTSD and depression have been shown to share a strong link as approximately 30-50% percent of individuals with PTSD also meet criteria for major depressive disorder (Nixon et al., 2004).

The model also highlights how increased anxiety can exacerbate one's experience of pain. As anxiety is the core proponent of PTSD, we can illustrate that PTSD alone increases the individual's perceived pain level. Sharp and Harvey also describe this process more specifically when they state that the intrusive memories that accompany PTSD trigger a very significant anxiety response. This anxiety response is often associated with increased muscle tension, which, for individuals with chronic pain, can elicit substantial pain responses.

The final factor identified in Sharp and Harvey's model is cognitive attention. They discuss how both chronic pain and PTSD are associated with high levels of cognitive activity.

The compounded cognitive demands that pain and PTSD command leave little cognitive capacity for individuals to utilize adaptive cognitive strategies, which are often critical components of the evidence-based psychological treatments for both PTSD and chronic pain (CPT and CBT-CP). Individuals with chronic pain and PTSD often develop catastrophic thought patterns that significantly impact their psychological well-being. While it is often easy to identify catastrophic thoughts and fairly straightforward to treat them, individuals with PTSD and chronic pain are left with a diminished capacity to utilize cognitive treatment approaches.

A more recent study by Liedl and colleagues (2010) aimed to further examine the underpinnings of the mutual maintenance model and establish a more empirical basis for its seven components. One of the most prominent findings from their study was the establishment of causality between arousal and the development of pain. Liedl and colleagues add that high arousal was identified as a core mechanism in the persistence or chronicity of pain. Additionally, Liedl and colleagues investigated the component of the model that suggests pain serves as a reminder of the trauma. Their results indicated that the experience of a trauma often resulted in increased activity the central and autonomic nervous systems as well as the musculoskeletal system. They argue that this activation continues as the individual displays symptoms of hypervigilence (i.e. muscle tension, insomnia, exaggerated startle response), which ultimately maintains and even escalates an individual's pain level. Liedl and colleagues build upon this information and suggest that hypervigilence symptoms and pain mutually maintain each other via a feedback loop. More specifically, they suggest that hypervigilence increases pain which increases hypervigilence signifying a perpetual relationship. Lastly, Liedl and colleagues suggest that because traumatic memories have been shown to alter perceptive mechanisms in the brain

(Moores et al., 2008) that they may also impact the way in which an individual interprets somatic symptoms such as pain.

Shared vulnerability model. Building upon the foundation of the mutual maintenance model, Asmundson and colleagues (2002) present the shared vulnerability model of co-occurring PTSD and chronic pain. They postulate that the link between chronic pain and PTSD is likely related to specific traits that predispose individuals to both the development of chronic pain as well as PTSD following a traumatic event. One of these specific traits that Asmundson and colleagues highlight is anxiety sensitivity. Anxiety sensitivity is often described as a fear of experiencing anxiety in the anticipation that it may have harmful consequences. It is important to note that anxiety sensitivity is not dichotomous in that all individuals fall somewhere on the spectrum from high to low levels of anxiety sensitivity. Asmundson and colleagues argue that a higher degree of anxiety sensitivity leaves individuals vulnerable for the development of PTSD as well as chronic pain after they have experienced a traumatic event. Similar to anxiety sensitivity, Asmundson and colleagues argue that if individuals experience a greater perception of lack of control at baseline function, then they are more likely to develop both conditions. They discuss the way in which both sensations of pain and traumatic experiences are largely uncontrollable events, which ultimately compounds individuals' already high levels of perceived uncontrollability.

Additionally, Asmundson and colleagues identify that individuals with a lower threshold for sympathetic nervous system activation may be predisposed to develop both chronic pain and PTSD following a traumatic event. They describe that often these individuals will be more distraught about and assume catastrophic implications for symptoms of sympathetic nervous system activation (i.e. heart racing, nausea, dizziness). Lastly, Asmundson and colleagues note

that selective attention for threat serves as a vulnerability factor for individuals to develop PTSD and chronic pain following a traumatic event. Globally, the shared vulnerability model posits that each of these identified factors cumulate to provide a greater degree of vulnerability for the individual to develop both PTSD and chronic pain.

The diathesis-stress model of chronic pain following traumatic injury. The diathesis-stress model of chronic pain (Turk, 2002) offers insight into the psychological complexities of chronic pain following a traumatic injury. Turk illustrates the psychological component of pain by stating that there are individuals who experience injury and develop chronic pain that eventually resume their usual activities and responsibilities while at the same time, there are individuals in the same scenario who do not return to normal levels of functioning. Similar to the shared vulnerability model, Turks's diathesis-stress model hypothesizes that individuals who are more emotionally reactive (diathesis) are more likely to develop avoidant and maladaptive responses following a traumatic event (stress). More specifically, the model postulates that a traumatic injury will induce greater disability in individuals who display a specific set of characteristics. One of these characteristics is best described as a tendency to interpret negative bodily symptoms as indicative of a serious problem or condition. Turk identifies this characteristic as stemming from maladaptive cognitive processing in where the individual makes an appraisal of the situation largely based on assumption and fear. He describes that, following a trauma, these individuals tend to interpret physical sensations as abnormal, harmful, and painful, which ultimately increases their anxiety. He illustrates how a different, more positive appraisal of the situation, such as "pain is a normal part of healing", would induce much less distress.

Additionally, Turk identifies that a preoccupation with bodily processes can also increase the likelihood of greater disability following a traumatic event. He notes that a preoccupation

with bodily processes is most often associated with anxiety sensitivity, which heightens the individual's response to anxiety-provoking stimuli and often increases their awareness to threatening stimuli or situations. Turk also postulates that certain individuals are predisposed to respond negatively and fearfully when contemplating the implications of perceived symptoms.

Lastly, Turk ascertains that the belief that avoidance of any potentially painful activity is the way to avoid further exacerbation of pain and disability also increases one's likelihood of developing greater disability after a traumatic injury. Turk discusses several negative consequences to the avoidance of activity. First, he illustrates that this type of avoidance prevents the individual from acquiring any corrective feedback to discount their beliefs about re-injury. The avoidance patterns also greatly impact their physical condition as their lack of activity leads to muscle deterioration and decreased mobility. Additionally, Turk discusses how the increased patterns of inactivity confirm the individual's self-identified belief that he or she is disabled. Lastly, Turk notes that as the avoidance tendencies become stronger, the individual experiences less perceived control over their symptoms leading to greater distress and disability.

In addition to identifying these "at risk" characteristics, the model attempts to highlight the specific role that trauma plays in the development of chronic pain. Following a traumatic event, individuals often fear that they are seriously injured and, even without confirming any injury, will begin to fear for eventual disability and plan for avoidance of potentially harmful stimuli (Crombez et al., 1999). It has been noted that maladaptive fear-avoidance beliefs in patients with chronic pain are of particular importance when the patient's pain originates from a traumatic injury as these beliefs tend to be more rigid and rooted in deeper anxiety (Turk & Rudy, 1986). Traumatic injury has also been shown to effect an individual's perception of pain. Lee and colleagues (1993) found that individuals who had been in motor vehicle accidents

reported significantly lower pain tolerances compared to healthy individuals. Trauma also appears to impact the treatment of chronic pain. Turk and Okifuji (1996) note that individuals who attribute their pain to a physical trauma were significantly more likely to be treated with more physical treatments such as nerve blocks or physical therapy. Additionally, Turk and Okifuji note that these individuals with traumatic-onset pain were five times more likely to receive an opioid prescription than those with a non-traumatic onset pain, even without evidence of greater physical pathology. The diathesis-stress model clearly articulates the ways in which psychological factors, such as anxiety sensitivity and cognitive processes, significantly affect an individual's experience of pain, level of disability, and recovery.

Treating chronic pain and posttraumatic stress disorder together. The literature base on the chronic pain and PTSD dual diagnosis has only begun to flourish and as such, the treatment literature for this dual diagnosis is limited. However, some of the initial treatment literature highlights specific treatment considerations and recommendations. One of the most prevalent findings within the treatment literature for PTSD and chronic pain is the need for a comprehensive treatment that addresses both conditions simultaneously. A host of relevant literature illustrates the inadequacy of individual approaches to these conditions. Farmer, Morone, and Karp (2009) found that pain severity that lead to interference of daily activities had a significant negative impact on an individual's response to anxiety treatment. Moreover, Sharp and Harvey (2001) note that because pain utilizes a great deal of cognitive demand, individuals' abilities to engage in evidence-based cognitive interventions for PTSD, such as cognitive processing therapy, is likely significantly diminished. Roth and colleagues (2008) support the need for comprehensive treatments in identifying that increased attention should be placed on PTSD treatment in individuals with both PTSD and chronic pain when chronic pain and

depression treatment efforts have been disappointing. They note that this disappointing result is often indicative of the "anchoring" role that PTSD symptoms can have on pain-related disability and distress. Additionally, it has been identified that patients with a comorbid history of trauma demonstrate a significantly reduced response to chronic pain treatment when compared to patients without trauma history (Hickling & Blanchard, 1992). Clapp and colleagues (2008) found that pain severity and emotional numbing symptoms of PTSD together impacted both role functioning and life satisfaction. They go on to suggest that interventions for individuals with both chronic pain and PTSD should target both physical pain and emotional numbing in an effort to significantly improve their quality of life.

Asmundson (2014) also supports the need for a comprehensive treatment approach by suggesting that it is insufficient to treat only PTSD or only chronic pain in individuals who present with both. He proposes two suggestions; one being that an integrated, comprehensive treatment approach is developed to accurately address chronic pain and PTSD simultaneously. Asmundson notes that this would likely required a multidisciplinary approach in which providers form integrated health teams and work collaboratively to address both of the patient's conditions. Asmundson's alternative suggestion is that providers and healthcare organizations dedicate resources to identifying and addressing shared vulnerability factors either prior to or at the beginning of the development of these conditions as a means to short-circuit their mutual maintenance properties.

Finucane and colleagues (2012) also offer insight into the treatment of chronic pain and PTSD. In their analysis of basic emotion profiles in individuals with chronic pain, PTSD, depression, and a healthy control group, Finucane and colleagues identified that anxiety about the experience of negative emotions is common to many psychological disorders as well as a

variety of health conditions. They discuss the particular importance of this for individuals with chronic pain and PTSD. They suggest that interventions for this population should target the anxiety of experiencing negative emotions and aim to reduce fear experiences in these individuals, mainly through a wide variety of exposure experiences.

Depression

Depression and posttraumatic stress disorder. Depression is one of the most prevalent and well-documented comorbidities of PTSD. It is estimated that between 30-50% of individuals with PTSD also meet criteria for major depressive disorder (Nixon et al., 2004; Hankin et al., 1999). A study done by Frueh and colleagues on veterans of the Vietnam era found that, of the individuals seeking treatment for PTSD, approximately 88% also had co-morbid depression. Campbell and colleagues (2007) note the positive relationship between PTSD symptom severity and the prevalence of suicidal ideation, which is increased when the individual also meets full criteria for major depressive disorder (Campbell et al., 2007). Turvey and colleagues (2002) state that PTSD with a co-morbid major depressive disorder diagnosis presents additional suicide risk factors for an individual through decreased social support and greater actual or perceived disability. It has been hypothesized that PTSD and depression share neurobiological abnormalities that foster or predispose the development of both conditions (Scioli-Salter et al., 2015). The PTSD and major depression dual-diagnosis also presents implications for treatment. In their study of individuals with PTSD and depression in primary care settings, Hegel and colleagues (2005) found that individuals with PTSD and depression experienced a significantly delayed response to a primary care depression intervention in comparison to individuals who presented with only major depression.

Depression and chronic pain. As noted, chronic pain and depression are undeniably correlated with one another. Chronic pain has been linked to higher rates of major depression, suicidal ideation, and suicide attempts (Ratcliffe et al., 2008). It is estimated that between 40% and 60% of patients with chronic pain also have depression (Bair et al., 2003; Kroenke et al., 2009). In addition to the evidence regarding the co-occurrence of depression and chronic pain, a portion of the literature describes more impactful and mechanistic aspects of the relationship. Demyttenaere and colleagues (2006) illustrated the significant impact that depression had on self-reported levels of pain, specifically noting that the presence of depression in chronic pain patients lead to reports of greater perceived pain. Conner and colleagues (2006) support this relationship with their finding that depression history increases the likelihood of increased pain perception. Additionally, Blackburn-Munro and Blackburn-Munro (2001) propose a common neurophysiological mechanism for co-occurring chronic pain and depression in discussing the effects these conditions have on the hypothalamic-pituitary-adrenal (HPA) axis. They discuss the way in which both chronic pain and depression serve as persistent and relentless stressors, which chronically activates the HPA axis and produces a long-term stress response involving both biological and psychological consequences.

Finucane and colleagues (2012) also shed light on the relationship between depression and chronic pain in their analysis of basic emotional profiles across individuals with depression, PTSD, or chronic pain as well as healthy individuals. They found that both the depressed group and the chronic pain group exhibited and reported a significantly fewer happiness experiences and higher rates of sadness. While these emotional profiles were anticipated in the depressed group, their presence in the chronic pain group represented the high prevalence of depressive symptoms within individuals with chronic pain. Finucane and colleagues highlight a finding by

Zautra and colleagues (2005) that identifies the role that positive affects plays in building pain resilience, primarily by mitigating the relationship between chronic pain and negative affect. Based upon these findings and their own results, Finucane and colleagues propose that treating depression in chronic pain patients should ultimately increase positive affect, build resilience to pain and decrease overall distress.

Depression, posttraumatic stress disorder, and chronic pain. As illustrated, both chronic pain and PTSD have been linked to depression as distinct diagnoses. However, there is a small facet of the literature that illustrates the relationship between all three conditions. Toomey and colleagues (1994) explored the role of trauma in the development of depression in pain patients. They found that patients whose pain presented following a trauma had significantly higher rates of depressive symptoms than patients whose pain was idiopathic. Similarly, Turk and Okijuji (1996) found that while 55% of idiopathic pain patients in their sample reported depressive symptoms, 70% of patients with traumatic-onset pain in their sample reported depressive symptoms. It is clear that depression is significantly related to both PTSD and chronic pain and as such serves as a shared symptom between PTSD and chronic pain.

Summary

Both PTSD and chronic pain have received a wealth of attention in their respective areas of the literature. PTSD and chronic pain both demonstrate a significant prevalence within the general population and an even greater prevalence in the veteran population. Approximately 20% of veterans experience PTSD throughout their lifetime (Ramchand et al., 2010) and nearly 55% of veterans develop some form of chronic pain (National Institute of Health, 2015). Additionally, both PTSD and chronic pain pose significant impacts on healthcare costs and utilization (Toblin et al., 2010; Deykin et al., 2001).

As noted, the literature on the specific relationship these two conditions is quite limited. While there are a few existing theories on the relationship between PTSD and chronic pain, the empirical support in this domain is lacking significantly, particularly with respect to treatment of the dual-diagnosis condition. This study aims to shed light on not only the relationship between these two diagnoses but also the potential treatment implications their comorbidity poses.

Chapter III

Methods

This study was designed to examine treatment outcomes of CPT, in veterans with PTSD, with or without chronic pain, and with or without pharmacological treatment for pain, in the reduction of both PTSD and depression symptoms, utilizing the Posttraumatic Stress Disorder Checklist (PCL) and Beck Depression Inventory, Second Edition (BDI-II). Patient-level variables, including biological sex, race/ethnicity, and branch of service, were also included in analysis to further explore the relationship between certain demographics and PTSD and depression symptom reduction. Data were collected retrospectively via chart review.

Participants

The sample was comprised of 94 veterans (74 men and 20 women), all with a diagnosis of PTSD and completion of CPT in the VA Healthcare System. Demographic information and descriptive statistics for the sample are outlined in Table 1. The sample consists of three distinct cohorts, reflecting three unique categories of chronic pain status. Cohort 1 (n = 33) represents veterans who have a diagnosed chronic pain condition, and were treated with opioid medication for chronic pain during the course of CPT. Cohort 2 (n = 30) is comprised of veterans with a diagnosed chronic pain condition who were not treated with opioid medication, or any other observable treatment method, for chronic pain during the course of CPT. Cohort 3 (n = 31) represents veterans without a diagnosed chronic pain condition and without opioid medication during course of CPT.

Participant data were retrieved from a national VA database, with data originating from VA Medical Centers and Community Based Outpatient Clinics in various locations within the United States, providing a degree of diversity in demographic characteristics. As shown by Table

2, roughly three quarters of the total sample were men (n = 74; 78.7%), with the remaining 20 (21.3%) participants being female. This distribution was largely maintained across the three cohorts with the percentage of male participants ranging from 75.8% (Cohort 1) to 83.3% (Cohort 2). The mean age of participants in the total sample was 50.17 years old (SD = 13.35), with the age of participants ranging from 24 to 88 years old. Sixty-five (69.1%) participants identified as White or Caucasian, 17 (18.1%) participants identified as Black or African American, 6 (6.4%) participants identified as Hispanic or Latino, 5 (5.3%) participants identified as American Indian or Alaska Native, and 1 (1.1%) participant identified as Asian. Roughly half of the participants were married (n = 49; 52.1%), with 38 (40.4%) participants who were divorced or separated, 4 (4.3%) participants who were widowed, and 3 (3.2%) participants who were never married.

In terms of comorbid psychiatric conditions, approximately half of the participants (n = 53; 56.4%) had a comorbid depressive disorder, 15 (16%) had a comorbid anxiety disorder, 34 (36.2%) had a comorbid substance use disorder, and 11 (11.7%) had a comorbid sleep disorder. Over half of participants in the sample were prescribed an antidepressant medication (n = 52; 55.3%) while roughly one fourth of participants were prescribed a benzodiazepine medication (n = 21; 22.3%). This distribution was largely consistent within cohorts, with a few exceptions. First, cohorts appeared to differ considerably on prevalence of comorbid sleep disorders, with Cohort 1 (veterans with a chronic pain condition utilizing pharmacological treatment for pain) demonstrating the lowest prevalence (n = 1; 3%) and Cohort 2 (veterans with a chronic pain condition not utilizing pharmacological treatment for pain) demonstrating the highest prevalence (n = 6; 20%). Second, antidepressant medication use appears to be varied between cohorts with 51.5% (n = 17) of Cohort 1 utilizing antidepressant medication, 66.7% (n = 20) of Cohort 2

utilizing antidepressant medication, and 48.4% (n = 15) of Cohort 3 utilizing antidepressant medication. Finally, a mixed distribution was seen in benzodiazepine medication use between the three cohorts; Cohort 1 (n = 11; 33.3%), Cohort 2 (n = 4; 13.3%), and Cohort 3 (n = 6; 19.4%).

Table 1. ***Demographics of sample and descriptive statistics.*** **(n = 94)**

Variable	*n*	*%*	*M*	*SD*
Biological Sex				
Male	74	78.7		
Female	20	21.3		
Age (years)			50.17	13.35
Race/Ethnicity				
White/Caucasian	65	69.1		
Black/African American	17	18.1		
Hispanic/Latino	6	6.4		
American Indian/Alaska Native	5	5.3		
Asian	1	1.1		
Marital Status				
Divorced or separated	38	40.4		
Married	49	52.1		
Never married	3	3.2		
Widowed	4	4.3		
Branch of Service				
Army	55	58.5		
Navy	5	5.3		
Marines Corps	15	16		
Air Force	18	19.1		
Coast Guard	1	1.1		
Service Era				
WWII	1	1.1		
Korean War	1	1.1		
Vietnam	30	31.9		
Post-Vietnam	18	19.1		
Persian Gulf War	18	19.1		
OEF/OIF	26	27.7		
Combat Exposure				
No	63	67		
Yes	31	33		
Comorbid Psychological Conditions				
Depressive Disorder	53	56.4		
Anxiety Disorder	15	16		

Substance Use Disorder	34	36.2		
Sleep Disorder	11	11.7		
Psychiatric Medications				
Antidepressant	52	55.3		
Benzodiazepine	21	22.3		
Service Connection %				
0	9	9.6		
1- 49.9	13	13.8		
50 - 99.9	43	45.8		
100	29	30.9		
Number of CPT Sessions			11.89	1.21

Table 2. ***Demographics of sample and descriptive statistics by cohort*****. (n = 94)**

Variables	Cohort 1 *(chronic pain with medication)* *n = 33*		Cohort 2 *(chronic pain without medication)* *n = 30*		Cohort 3 *(no chronic pain)* *n = 31*	
	n	*%*	*n*	*%*	*n*	*%*
Biological Sex						
Male	25	75.8	25	83.3	24	77.4
Female	8	24.2	5	16.7	7	22.6
Age (years) *M*	*M* =	46.61	*M* =	48.40	*M* =	55.68
SD	*SD* =	12.75	*SD* =	14.84	*SD* =	10.86
Race/Ethnicity						
White/Caucasian	23	69.7	22	73.3	20	64.5
Black/African American	4	12.0	4	13.3	9	29
Hispanic/Latino	2	6.1	3	10	1	3.2
American Indian/Alaska Native	3	9.1	1	3.3	1	3.2
Asian	1	3	0	0	0	0
Marital Status						
Divorced or separated	11	33.3	11	36.6	16	51.6
Married	19	57.6	18	60	12	38.7
Never married	2	6.1	0	0	1	3.2
Widowed	1	3	1	3.3	2	6.5
Branch of Service						
Army	20	60.6	18	60	17	54.8
Navy	1	3	4	13.3	0	0
Marines Corps	6	18.2	5	16.7	4	12.9
Air Force	6	18.2	3	10	9	29
Coast Guard	0	0	0	0	1	3.2
Service Era						
WWII	0	0	1	3.3	0	0

Korean War		0	0	0	0	1	3.2
Vietnam		9	27.3	9	30	12	38.7
Post-Vietnam		7	21.2	3	10	8	25.8
Persian Gulf War		7	21.2	3	10	8	25.8
OEF/OIF		10	30.3	14	46.7	2	6.5
Combat Exposure							
No		21	63.6	17	56.7	25	80.6
Yes		12	36.4	13	43.3	6	19.4
Comorbid Psychological							
Depressive Disorder		19	57.6	17	56.7	17	54.8
Anxiety Disorder		7	21.2	4	13.3	4	12.9
Substance Use Disorder		12	36.4	9	30	13	41.9
Sleep Disorder		1	3	6	20	4	12.9
Psychiatric Medications							
Antidepressant		17	51.5	20	66.7	15	48.4
Benzodiazepine		11	33.3	4	13.3	6	19.4
Service Connection %							
0		4	12.1	3	10	2	6.5
1- 49.9		4	12.1	3	10	6	19.4
50 - 99.9		15	45.5	12	40	16	51.6
100		10	30.3	12	40	7	22.6
Number of CPT Sessions	*M*	*M* =	12.09	*M* =	11.70	*M* =	11.87
	SD	*SD* =	1.31	*SD* =	1.26	*SD* =	1.02

Procedures

Database. Data were extracted retrospectively from the VA Informatics and Computing Infrastructure (VINCI) database according to search parameters congruent with the population of interest; veterans with PTSD, with or without chronic pain and with or without pharmacological treatment for pain, who have completed CPT. VINCI was established through a partnership between the Veterans Health Administration's Office of Research and Development (VHA ORD) and the VA Office of Information Technology (OI&T) to provide a secure virtual workspace for access and collection of patient data in an effort to enhance clinical research. Populated administrative data was utilized for collection of certain demographic information, including race/ethnicity, marital status, and military service information. Additionally, chart reviews were

conducted to obtain additional demographic information, including age, information related to treatment, and confirmation of diagnoses.

Security of data. This study required use of protected health information such as medical and psychological diagnoses to identify eligible participants, social security numbers to access patients' charts, and dates of birth to calculate age. All protected health information was maintained on secure VINCI servers where only individuals directly associated with the project have access. No raw data was removed from the secure VINCI server nor was it made available to individuals outside of the study. The VINCI database ensures security of patients' protected health information, while providing more direct access to VA data in an effort to expand clinical research. Aggregated results from statistical analyses were dispersed beyond the VINCI server in an effort to disseminate overall findings and associated clinical implications.

Participant selection. Utilizing the VA Informatics and Computing Infrastructure (VINCI) data managers, three sub-pools were derived from a pool of veterans who had completed the PCL two or more times within a six-month period between January 1, 2006 and July 31, 2013. The first sub-pool further identified veterans with a diagnosed chronic pain condition and at least one outpatient opioid medication prescription. The second sub-pool was narrowed to veterans with a diagnosed chronic pain condition and no outpatient opioid medication prescriptions. The final sub-pool distinguished veterans without a diagnosed chronic pain condition and without any outpatient opioid medication prescriptions. Subsequently, veterans in each sub-pool were confirmed to have at least two PCL scores and two BDI-II scores to create the final sub-pools. The final set of sub-pools was comprised of 8929, with 1405 veterans in the first sub-pool, 4465 veterans in the second sub-pool, and 3059 veterans in the third-sub pool. From each sup-pool, participants were randomly selected for chart review.

Chart reviews were conducted using the Compensation and Pension Records Interchange (CAPRI). Of the 8929 veterans identified within the three sub-pools, 1007 veterans' charts were reviewed for assessment of inclusion and exclusion criteria. More specifically, 450 charts were reviewed from the first sub-pool, 246 charts from the second sub-pool, and 311 charts from the third sub-pool. These chart reviews resulted in a total sample size of 92 participants; 33 from the first sub-pool comprising cohort one, 30 from the second sub-pool resulting in cohort two, and 31 from the third sub-pool creating cohort three. Most commonly, veterans were excluded for PCL or BDI-II measurements outside of the identified pre and post treatment periods. Additionally, a substantial portion of veterans were excluded for incomplete engagements in CPT. There were also veterans excluded from particular cohorts based on disqualifying diagnoses or medications (e.g. veterans reviewed for Cohort 3 that had a chronic pain condition listed or were taking an opioid medication during time of treatment).

Chart reviews were utilized to ensure completion of CPT with assessment of PTSD and depression symptoms, utilizing the PCL and BDI-II, respectively. Only pre-treatment assessments that occurred within the four weeks prior or two weeks after the start of CPT were included. Similarly, only post-treatment assessments that occurred within the four weeks following the conclusion of CPT were included in an effort to promote internal validity. Only data from participants' initial regimen of CPT was included. Chart review was also utilized to verify alignment of chronic pain diagnoses and opioid medication status, as applicable.

To provide an accurate reflection of the overall population of veterans, exclusion criteria was limited and utilized only to reduce extraneous treatment variables. Participants were excluded if they simultaneously completed another individual psychological treatment for any mental health condition. Similarly, an additional, concurrent group therapy that addresses PTSD

or chronic pain specifically excluded participants. In terms of pain treatment, participants were excluded if there was a noted concurrent, alternative treatment for pain, which include acupuncture, physical therapy, and recreational therapy services. Additionally, data from subsequent regimens of CPT was not be included to avoid compounded treatment results. Data from CPT regimens with greater than sixteen sessions were also be excluded from the study, as additional sessions could provide added treatment effects and are less likely to resemble manualized CPT.

Ethical Issues

It is imperative to address and manage ethical issues in all forms of research, including the present study. As data was collected retrospectively, from VA Medical Centers and Community Based Outpatient Clinics throughout the United States, the primary ethical concerns included patient privacy, primarily with respect to confidentiality of personal health information, and autonomy as it relates to informed consent. The aforementioned data security measures were utilized in an effort to minimize risks to participants by maintaining confidentiality of personal health information.

This study was granted an exemption for informed consent given the minimal level of risk to participants with identified data security measures. Additionally, there is concern that contacting participants directly to obtain informed consent could result in further breaches of confidentiality by identifying participants' involvement in mental health services. Of note, all veterans utilizing VA services consent to their data being utilized in research. To ensure protection of participant information, all procedures and ethical issues were approved by the Internal Review Board (IRB) of the VA Eastern Kansas Healthcare system. Additionally this study, including information regarding risk to participants, security measures, and anticipated

benefits of the present study were also submitted to and approved by the University of Kansas Human Subjects Committee of Lawrence.

Power Analysis

In utilizing Cohen's (1988) parameters of effects sizes for F tests (small: 0.1 – 0.24; moderate: 0.25 – 0.4; and large: 0.4 or greater), use of the PCL in CPT outcome research, has demonstrated a moderate to large effect in the literature across multiple studies in various populations (Monson et al., 2006; Chard & LaGreca, 2005; Resick et al., 2002). Given this range reported, the power analysis explored conservative approaches to both a moderate and large effect size, providing a range for ideal sample size.

In utilizing G-Power version 3.1.9.4 (Faul, Erdfelder, Lang, & Buchner, 2009) to calculate an a-priori power analysis, results indicate a hypothetical study with three active treatment groups and two measurements over time would have a 95% chance of finding a true effect size of .25 to .45 if the sample contains 251 to 80 participants, respectively. The study aimed to obtain a total sample size near the most conservative margin, in an effort to account for comparison of treatment outcomes across three active treatment groups. However, due to availability of qualified data, a smaller sample size was obtained. Data availability appears to have been significantly impacted by exclusion criteria used. It will be a technical limitation of the study, but a decision was made to use the exclusion criteria to protect internal validity. As this study is to serve as a preliminary examination of group differences in CPT outcomes, the sample size is believed to allow meaningful inferences made with reasonable confidence.

Independent Variable Operationalization

Chronic pain status. Chronic pain status was determined using both diagnostic and treatment information resulting in three distinct levels: (1) chronic pain diagnosis with

pharmacological treatment for pain (identified via outpatient opioid prescription medication), (2) chronic pain diagnosis without pharmacological treatment for pain (identified via the absence of outpatient opioid prescription medication), and (3) no chronic pain diagnosis with no pharmacological treatment for pain (identified via the absence of outpatient opioid prescription medication). Both generalized and site-specific chronic pain conditions were included in this study. For this study, a pain condition was categorized as chronic in one of three ways. The first involved use of "chronic" in the diagnostic description and subsequent coding (e.g. "chronic low back pain" or "chronic pain syndrome"). Secondly, certain pain conditions were considered inherently chronic given the often unknown etiology, limited utility of diagnostics, and particularly undefined treatment options (e.g. Fibromyalgia). Lastly, pain conditions were identified as chronic if the chart reflected active persistence of a pain diagnosis for greater than six months, the most conservative metric of the duration criteria for chronic pain (International Association for the Study of Pain, 1986).

Dependent Variable Assessments

PTSD symptom severity change. The primary dependent variable in this study was the change in PTSD symptoms severity over the course of CPT. To assess the severity of PSTD symptoms, the Posttraumatic Stress Disorder Checklist (PCL; Weathers et al., 1993) was used. As the PCL was administered prior to and after CPT treatment, it serves as a repeated measure for the study The PCL has been empirically validated through over a dozen studies and is the most widely used self-report measure of PSTD symptom severity (McDonald & Calhoun, 2010; Wilkins, Lang & Norman, 2011). It has demonstrated high internal consistency ($\alpha = .97$) and strong test-retest reliability ($r = .96$) over a one-week period (Weathers et al., 1993). The PCL includes 17 self-report items in a questionnaire format that model the diagnostic criteria for

PTSD, as provided by the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV). Although the PCL offers a new edition (PCL-5) that models criteria from the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5), the retrospective data used for this study will align with the DSM-IV and therefore utilizes the original version of the PCL. The PCL asks individuals to rate the severity of specific PTSD symptoms, outlined in the DSM-IV, over the last month using a 5-point Likert scale. Total scores on the PCL range from 17-85 and indicate total symptom severity with lower scores reflecting fewer and less severe symptoms and higher score reflecting more symptoms with a greater severity. The PCL also offers three subscales, re-experiencing, avoidance/numbing, and hyperarousal, which reflect three of the major symptom clusters of PTSD. The PCL will have been administered once to participants prior to or at the beginning of treatment and once to participants after concluding therapy. Pre-treatment PCL scores collected within the four weeks prior or two weeks after the start of CPT were included. Similarly, only post-treatment PCL scores collected within four weeks of concluding CPT were included.

Depression symptom severity change. Additionally, change in depression symptom severity over the course of CPT was used as a dependent variable in this study. To assess the presence and severity of depressive symptoms, the Beck Depression Inventory, Second Edition (BDI-II; Beck et al., 1996) was utilized. As the BDI-II was administered prior to and after CPT treatment, it serves as a repeated measure for the study. The BDI-II is one of the most frequently used measures of depression and has been empirically validated across a variety of different populations, including veterans (Bringmann et al., 2015). It has demonstrated high internal consistency ($\alpha = .92$) and strong test-retest reliability ($r = .93$) over a one-week period (Beck et al., 1996). The BDI-II is a 21-item, self-report questionnaire that measures depression symptoms

presence and severity over the last two weeks, utilizing a 4-point Likert scale. Total scores on the BDI-II range from 0 to 63 with higher scores indicating a more severe presence and severity of depressive symptoms. The BDI-II offers total score ranges that reflect a specific degree of severity in symptoms. Scores ranging from 0-13 reflect minimal depression, scores from 14-19 reflect mild depression, scores from 20-28 reflect moderate depression, and scores over 29 reflect severe depression (Beck et al., 1996). BDI-II scores were collected in the same timeframe as the PCL with the pre-treatment score obtained within the four weeks prior or two weeks after the start of CPT and the pos-treatment score obtained within the four weeks following the conclusion of CPT.

Research Questions and Hypotheses:

Research Question I: Are there significant differences between pre- and post-CPT measurements of PTSD symptom severity?

Hypothesis I: There are significant differences between pre- and post-CPT measurements of PTSD symptom severity, regardless of veterans' chronic pain status.

Research Question II: Are there significant differences between pre- and post-CPT measurements of depression symptom severity?

Hypothesis II: There are significant differences between pre- and post-CPT measurements of depression symptom severity, regardless of veterans' chronic pain status.

Research Question III: Are there significant differences in the degree of PTSD symptom change, between three cohorts of veterans: 1) veterans with a chronic pain condition and pharmacological treatment for pain, 2) veterans with a chronic pain condition without pharmacological treatment for pain, and 3) veterans with no chronic pain condition and no pharmacological treatment for pain, following engagement in CPT?

Hypothesis III: There are significant differences in the degree of PTSD symptom change between the three cohorts of veterans: 1) veterans with a chronic pain condition and pharmacological treatment for pain, 2) veterans with a chronic pain condition without pharmacological treatment for pain, and 3) veterans with no chronic pain condition and no pharmacological treatment for pain, following engagement in CPT, with Cohort 2 demonstrating significantly less PTSD symptom change than Cohorts 1 and 3.

Research Question IV: Are there significant differences in depression symptom change, between the three cohorts of veterans: 1) veterans with a chronic pain condition and pharmacological treatment for pain, 2) veterans with a chronic pain condition without pharmacological treatment for pain, and 3) veterans with no chronic pain condition and no pharmacological treatment for pain, following engagement in CPT?

Hypothesis IV: There are significant differences in depression symptom change between the three cohorts of veterans: 1) veterans with a chronic pain condition and pharmacological treatment for pain, 2) veterans with a chronic pain condition without pharmacological treatment for pain, and 3) veterans with no chronic pain condition and no pharmacological treatment for pain, following engagement in CPT, with Cohort 2 demonstrating significantly less depressive symptom reduction than Cohorts 1 and 3.

Data Analysis

Preliminary analyses. Preliminary analyses included descriptive analyses of the sample's demographic and treatment-related variables to provide an overview of the dataset. Of particular interest were the demographic variables of age, gender, race/ethnicity, and marital status. Additionally, other patient-level and treatment-related variables such as branch of service, war era, combat exposure, comorbid psychiatric conditions, psychiatric medications and number

of sessions required to complete CPT. Bivariate correlational analyses (for continuous variables) and independent samples *t*-tests (for dichotomous variables) were conducted to identify covariates of interest by assessing significance in the relationship between the outcome variables (post-treatment PCL scores and post-treatment BDI-II scores) and the demographic, other patient-level, and treatment-related variables and outcome variables.

Inferential analysis. To test the hypotheses of the study a multivariate analysis of variance (MANOVA) with repeated measures was utilized. More specifically, a 2 (time of measurement: pre-treatment, post-treatment) X 3 (group: no chronic pain, chronic pain and no pharmacological treatment, and chronic pain and pharmacological treatment) MANOVA was conducted, using the outcomes of PTSD and depression symptom scores measured with the PCL and the BDI-II as dependent variables. MANOVA with repeated measures was selected to account for the unique variance of each variable while limiting experiment-wise error and maximizing statistical power. MANCOVA was not utilized, as no significant covariates were identified through the correlational analysis.

Post-hoc analyses. Fisher's Least Significant Difference (LSD) post-hoc analysis was utilized to more specifically delineate significant between-group differences.

Chapter IV

Results

Two primary research questions guided both the initial design and subsequent analyses for this study. First, this study aimed to explore changes in posttraumatic stress disorder (PTSD) and depression symptom severity following a regimen of cognitive processing therapy (CPT), across all cohorts in the sample. Second, this study explored distinctions in degree of symptom change following CPT for both PTSD and depression symptoms between three cohorts, all of whom were diagnosed with PTSD:

1) Veterans with a chronic pain condition who were engaged in pharmacological treatment for pain
2) Veterans with a chronic pain condition who were not engaged in pharmacological treatment for pain
3) Veterans who do not have a chronic pain condition and were not engaged in pharmacological treatment for pain

Preliminary Analyses

Covariates were to be included in the model after empirical examination for association with either outcome of interest (PCL post-treatment score and BDI-II post-treatment score). Various demographic variables were explored for inclusion as covariates utilizing independent samples *t*-tests (if covariate was dichotomous) and bivariate correlations (if covariate was continuous). Race was not associated with PCL post-treatment score ($t(92) = -0.92$, $p = 0.36$) or BDI-II post-treatment score ($t(92) = -0.53$, $p = 0.60$). Biological sex was not associated with PCL post-treatment score ($t(92) = -0.38$, $p = 0.71$) or BDI-II post-treatment score ($t(92) = -1.48$, $p = 0.14$). Age was not significantly correlated with PCL post-treatment score ($r = -0.06$, $p = 0.56$) or

BDI-II post-treatment score (r = -0.12, p = 0.27). Marital status was also not associated with PCL post-treatment score ($t(92)$ = 1.25 , p = 0.22) or BDI-II post-treatment score ($t(92)$ = 1.20, p = 0.23). As a result, no demographic covariates were included in MANOVA analyses.

Additionally, OEF/OIF service era veterans did not differ from other service era veterans on PCL post-treatment scores ($t(92)$ = -1.10, p = 0.27) or BDI-II post-treatment scores ($t(92)$ = -1.57, p = 0.12). Similarly, Veterans who served in the Army did not differ significantly from Veterans who served in other branches of the military on PCL post-treatment score ($t(92)$ = -0.41, p = 0.69) or BDI-II post-treatment score ($t(92)$ = -0.63, p = 0.53). Combat exposure was not significantly related to either PCL post-treatment score ($t(92)$ = -1.06, p = 0.29) or BDI-II post-treatment score ($t(92)$ = -1.28, p = 0.21). Antidepressant medication use was not associated with PCL post-treatment score ($t(92)$ = -1.48, p = 0.14) or BDI-II post-treatment score ($t(92)$ = -0.53 , p = 0.60). Additionally, benzodiazepine medication use was not associated with PCL post-treatment score ($t(92)$ = 0.62, p = 0.54) or BDI-II post-treatment score ($t(92)$ = 0.57, p = 0.57). Associations between several co-morbid psychological conditions and outcome measures (PCL post-treatment scores and BDI-II post-treatment scores) were examined with no significant correlations observed: comorbid depressive disorder (PCL: $t(92)$ = 0.65, p = 0.52; BDI-II: $t(92)$ = -0.30, p = 0.77), comorbid anxiety disorder (PCL: $t(92)$ = -0.19, p = 0.85; BDI-II: $t(92)$ = -0.07, p = 0.94), comorbid substance use disorder (PCL: $t(92)$ = 0.99, p = 0.33; BDI-II: $t(92)$ = 1.42 , p = 0.16) and comorbid sleep disorder (PCL: $t(92)$ = -1.13, p = 0.26; BDI-II: $t(92)$ = -1.60, p = 0.11). Finally, number of CPT sessions was not associated with either PCL post-treatment score (r = -0.05, p = 0.63) or BDI-II post-treatment score (r = 0.03, p = 0.79). As a result of the analyses, none of the examined covariates were included in the final MANOVA model. See Table 3 for results.

Table 3. *Significance Tests for Potential Covariates*

		PCL Post-Score	BDI-II Post-Score
Race	*t* =	-0.92	-0.53
	p =	0.36	0.60
Biological Sex	*t* =	-0.38	-1.48
	p =	0.71	0.14
Age	*r* =	-0.06	-0.12
	p =	0.56	0.27
Marital Status	*t* =	1.25	1.20
	p =	0.22	0.23
War Era	*t* =	-1.10	1.57
	p =	0.27	0.12
Branch of Service	*t* =	0.41	-0.63
	p =	0.69	0.53
Combat Exposure	*t* =	-1.06	-1.28
	p =	0.29	0.21
Antidepressants	*t* =	-1.48	-0.53
	p =	0.14	0.60
Benzodiazepines	*t* =	0.62	0.57
	p =	0.54	0.57
Depressive Disorder	*t* =	0.65	-0.30
	p =	0.52	0.77
Anxiety Disorder	*t* =	-0.19	-0.07
	p =	0.85	0.94
Substance Use Disorder	*t* =	0.99	1.42
	p =	0.33	0.16
Sleep Disorder	*t* =	-1.13	-1.60
	p =	0.26	0.11
Number of CPT Sessions	*r* =	-0.05	0.03
	p =	0.63	0.79

Inferential Analyses

To collectively test the hypotheses while accounting for the unique variance of each variable, a repeated-measures multivariate analysis of variance (MANOVA) was conducted. The

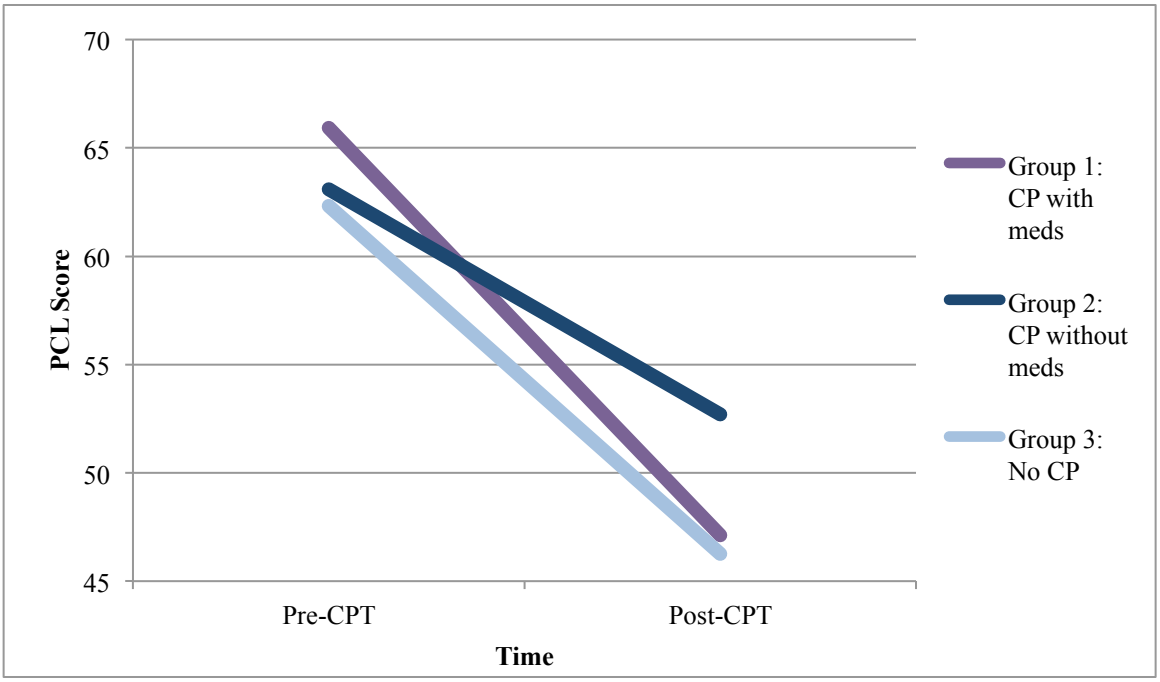
use of MANOVA also limited experiment-wise error and maximized statistical power. Furthermore, the high correlation observed between PTSD and depressive symptom scores in this sample ($r = 0.70$) necessitated a model that included both outcomes in a single analysis; thus, MANOVA was determined to be the most comprehensive, parsimonious analytic method. The MANOVA examined main effects of time and group on PTSD and depression symptom scores, as well as the time by group interaction.

Table 4 displays the results of the MANOVA analysis. As shown, significant within-group differences were demonstrated in both PTSD symptom severity change and depression symptom severity change. Additionally, between-group differences in depression symptom severity change were found to be significant. Specifically, the MANOVA revealed a main effect of time on PCL scores, $F(1, 93) = 110.8$, $p < .001$, $\eta_p^2 = 0.549$, suggesting significant change in PTSD symptoms from pre-treatment to post-treatment, across all three cohorts (pre-treatment: $M = 63.84$, $SD = 9.20$; post-treatment: $M = 48.62$, $SD = 17.17$). Similarly, a main effect of time on BDI-II scores was observed in the MANOVA, $F(1,93) = 76.9$, $p < .001$, $\eta_p^2 = .549$, suggesting significant depression symptom change, per self-report on the Beck Depression Inventory, Second Edition (BDI-II), across all three cohorts in the sample (pre-treatment: $M = 31.99$, $SD = 10.79$; post-treatment: $M = 21.65$, $SD = 12.90$). These main effects support Hypotheses I and II. Of note, the main effect of group on both PTSD symptom severity and depression symptom severity was non-significant (PCL: $F(2, 91) = 0.73$, $p = .487$; BDI-II: $F(2, 91) = 1.78$, $p = .174$). There were no hypotheses associated with this finding.

The MANOVA yielded a significant interaction effect of time by group on depression symptom change ($F(2, 91) = 5.463$, $p = .006$, $\eta_p^2 = .107$) but not on PTSD symptom change ($F(2, 91) = 2.96$, $p = .056$, $\eta_p^2 = .061$), although results appear to approach significance. Of note,

results indicated a low observed power (0.564) metric in the time by group interaction effect on the PCL, demonstrating potential for type II error; failure to observe significant effects. See Table 5 for group means and Figure 1 for illustration.

Figure 1. *Between-group differences in pre- and post-treatment PTSD symptoms.*



Note. "CP" = chronic pain. "Meds" = medication.

The significant interaction effect of time by group on depression symptom reduction supported Hypothesis IV. This interaction effect suggests differential symptom change in depression symptoms among the three cohorts: Veterans with PTSD and a chronic pain condition with pharmacological treatment for pain (pre-treatment: $M = 33.94$, $SD = 9.69$; post-treatment: $M = 20.42$, $SD = 12.91$), Veterans with PTSD and a chronic pain condition without pharmacological treatment for pain (pre-treatment: $M = 31.50$, $SD = 11.13$; post-treatment: $M = 26.73$, $SD = 13.42$), and Veterans with PTSD and no chronic pain condition (pre-treatment: $M = 30.39$, $SD = 11.57$; post-treatment: $M = 18.03$, $SD = 11.09$). See Table 5 for group means and Figure 2 for illustration.

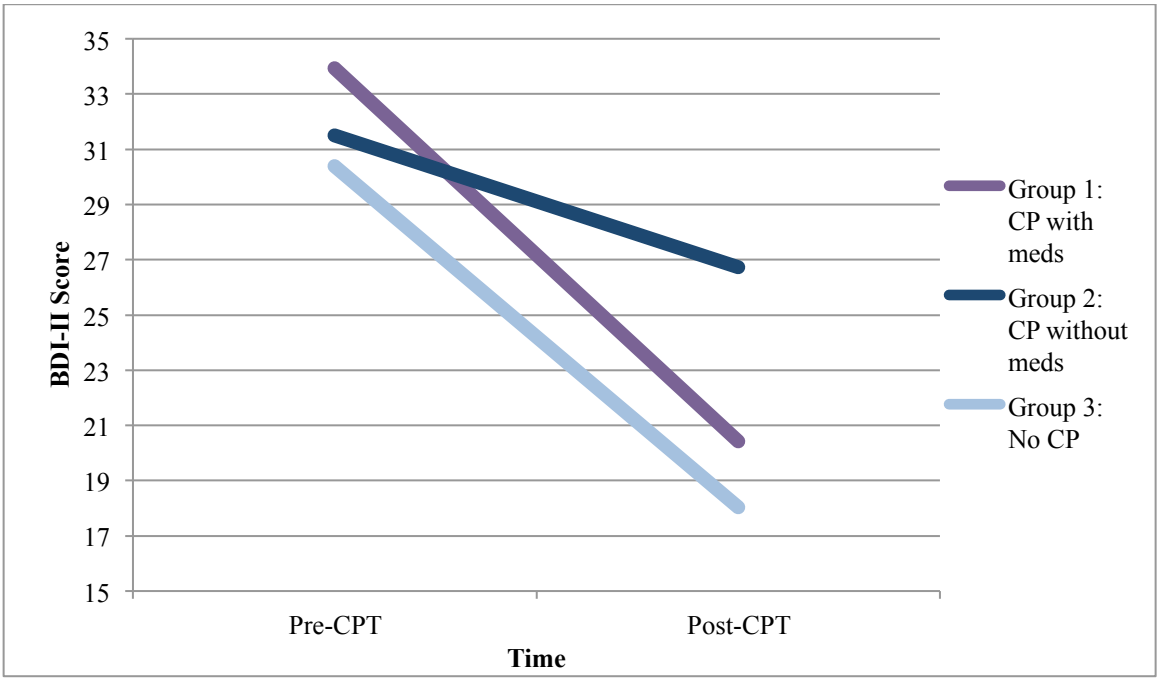
To identify specific differences between the cohorts in depression symptom change, while also obtaining statistical confidence in between-group differences, Fisher's Least Significant Difference (LSD) post-hoc analysis was performed. The analysis failed to yield significance, although the result approaches significance (p = .064). See Table 6 for results.

Table 4. *Within-Group and Between-Group Effects at Pre- and Post-Treatment*

		SS	*df*	*MS*	*F*	*Sig.*	*Partial* n^2
Time	PCL	10691.53	1	10691.53	110.76	.000	.549
	BDI-II	4893.95	1	4893.95	76.91	.000	.458
Group	PCL	406.55	2	203.28	.725	.487	.016
	BDI-II	747.58	2	373.79	1.784	.174	.038
*Time*Group*	PCL	573.16	2	286.58	2.97	.056	.061
	BDI-II	695.20	2	347.60	5.46	.006	.107

Note. Significance indicated with p<0.05.

Figure 2. *Between-groups differences in pre- and post-treatment depressive symptoms.*



Note. "CP" = chronic pain. "Meds" = medication.

Table 5. *Marginal Means for Time X Group Interaction*

			95% Confidence Interval	
		M	Lower Bound	Upper Bound
PCL	CP w/meds: *pre-treatment*	65.94	62.77	69.108
	CP w/meds: *post-treatment*	47.12	41.20	53.04
	CP w/out meds: *pre-treatment*	63.10	59.78	66.42
	CP w/out meds *post-treatment*	52.70	46.49	58.91
	No CP: *pre-treatment*	62.32	59.05	65.59
	No CP: *post-treatment*	46.26	40.15	52.37
BDI-II	CP w/meds: *pre-treatment*	33.94	30.21	37.67
	CP w/meds: *post-treatment*	20.42	16.10	24.75
	CP w/out meds: *pre-treatment*	31.50	27.58	35.42
	CP w/out meds *post-treatment*	26.73	22.20	31.27
	No CP: *pre-treatment*	30.39	26.54	34.24
	No CP: *post-treatment*	18.03	13.57	22.50

Table 6. *Fisher's Least Significant Difference for BDI-II*

			95% Confidence Interval	
	Mean Difference	*Sig.*	*Lower Bound*	*Upper Bound*
CP w/meds v. CP w/out meds	-1.93	.456	-7.06	3.19
CP w/meds v. No CP	2.97	.249	-2.11	8.06
CP w/out meds v. No CP	4.91	.064	-0.30	10.11

Note. Significance indicated with $p<0.05$.

Summary

In summary, the MANOVA analysis showed that self-reported PTSD and depressive symptom scores decreased from pre- treatment to post-treatment after application of CPT for the entire sample. Between-group differences were not observed on PCL scores. Moreover, the MANOVA revealed a significant time by group interaction on BDI-II scores. However, Fisher's Least Significance Difference post-hoc analysis failed to show significant group differences.

Chapter V

Discussion

This study was designed to explore the relationship between posttraumatic stress disorder (PTSD) and chronic pain by examining group differences in treatment outcomes following engagement in cognitive processing therapy (CPT) among three cohorts of veterans who differ on chronic pain status. More specifically, the study examined change in PTSD and depression symptom severity following CPT among veterans with or without chronic pain and with or without pharmacological treatment for pain. The goals of the study were two-fold; first, demonstrating preliminary evidence for potential effectiveness of CPT in this clinical sample and second, providing preliminary evidence that supplemental pain treatments are potentially beneficial in this comorbid population. Using the observational data, the study findings answered four research questions by testing four hypotheses. This chapter offers a summary and interpretation of the findings, while also discussing how these findings supplement the existing literature on PTSD and chronic pain. Additionally, limitations of the study, treatment implications, and future directions for research are discussed.

Summary and Discussion of Findings

Sample demographics and their association with major study variables. A diverse sample in three cohorts was obtained for the study. The analyses of the association between treatment outcomes (PCL and BDI-II post-treatment scores) and the following variables: race, biological sex, age, marital status, war era, branch of service, combat exposure, comorbid psychiatric medication use, comorbid psychological conditions and number of CPT sessions utilized to complete treatment, failed to show any significant results. Thus, no demographic variables were included in the final MANOVA model.

The average symptom presentation among the three cohorts was shown in the main effect of group in the MANOVA. Results indicated no significant differences in average PTSD and depression symptoms between the three cohorts before treatment: 1) Veterans with a chronic pain condition and pharmacological treatment for pain, 2) Veterans with a chronic pain condition without pharmacological treatment for pain, and 3) Veterans with no chronic pain condition and no pharmacological treatment for pain. This may represent similar pre-treatment symptom severities across the cohorts for both PTSD symptoms and depression symptoms.

Some of these non-significant results were intriguing in that the established literature provides contradicting evidence. For instance, the literature has shown that the presentation and treatment of PTSD may be associated with chronic pain, biological sex or gender, benzodiazepine medication use, and comorbid substance use disorders. As previously discussed in this paper, it has been theorized that chronic pain may perpetuate symptoms of PTSD, resulting in increased PTSD symptom severity (Sharp & Harvey, 2001; Leidl et al., 2010) and potential for PTSD treatment disturbance (Asmundson, 2014). The lack of significant differences in average symptom presentation among the three cohorts appears surprising given the support for distinction in the literature. Several studies have identified significant differences trauma exposure and presentation of PTSD symptoms between men and women (Tiet et al., 2015; Tolin & Foa, 2006). Certain studies have alluded to the notion that these differences impact treatment outcomes. For example, Tolin and Foa (2006) found that men tend to exhibit more symptoms of aggression, irritability, and other externalizing behaviors that often lead to poorer treatment outcomes. However, there is also a substantial portion of the literature that demonstrates non-significant relationships between gender or biological sex and PTSD treatment outcomes (Litz et al., 2010; Karatzias et al., 2007; Taylor et al., 2003). As such, the non-significant finding

between biological sex and CPT treatment outcomes observed in the present study blends with the mixed evidence in the existing literature.

Additionally, consideration of the negative effects of benzodiazepine medication use on exposure-based treatments, particularly in PTSD treatment, has been more recently explored in the literature (Lund et al., 2013; Hawkins et al., 2012). As such, the non-significant relationship between benzodiazepine use and CPT treatment outcomes appears to be somewhat surprising. Similarly, the relationship between PTSD and substance use has been well document in the literature (Frueh et al., 2005; Manhapra et al., 2015; Hein et al., 2010). Additionally, comorbid substance use has also been shown to complicated PTSD treatment (Stewart et al., 2002). Given these findings, the non-significant relationship observed in the present study between comorbid substance use disorders and treatment outcomes appears unexpected. It is possible that the inconsistent findings observed in the present study are a product of the limited size of the sample. As such, further exploration is needed.

Possible roles of cognitive processing therapy in posttraumatic stress disorder and depression symptom change among all three cohorts. Hypothesis I and II predicted that Veterans would demonstrate significant reduction PTSD symptoms and depression symptoms, respectively, from pre-treatment to post-treatment, regardless of chronic pain status, which was supported by the significant main effect of time on both PCL and BDI-II scores in the MANOVA. Although reduction in symptoms cannot be attributed to the treatment effects of CPT alone in this study, due to the lack of experimental design and absence of a control group, these results may warrant consideration that CPT is effective in reducing PTSD and depression symptoms for veterans with PTSD, regardless of chronic pain status. This positive outcome would indicate broad utility of CPT, including for veterans with chronic pain conditions. The

results from the present study compliment a host of existing literature surrounding the general efficacy and effectiveness of CPT in reduction of PTSD and depression symptoms (Monson et al., 2006; Foa et al., 2008; Haller et al., 2015). However, within this body of research, there is no identified study that has specifically examined the efficacy of CPT in a chronic pain population. It appears significant that a sample, comprised primarily of veterans with a chronic pain condition, showed significant reduction in PTSD and depression symptoms after receiving CPT treatment. Further, CPT efficacy research appears warranted for this population.

Differential degree of posttraumatic stress disorder and depression symptom change following cognitive processing therapy between the three cohorts. Hypothesis III predicted significant differences in the degree of PTSD symptom reduction between the three cohorts: 1) Veterans with a chronic pain condition and pharmacological treatment for pain, 2) Veterans with a chronic pain condition without pharmacological treatment for pain, and 3) Veterans with no chronic pain condition and no pharmacological treatment for pain, with Cohort 2 demonstrating significantly less PTSD symptom reduction than Cohorts 1 and 3. The MANOVA result failed to support this hypothesis The interaction effect of time by group on PCL scores in the MANOVA did not reach the pre-set statistical significance level at $p < .05$, however, did appear to approach significance ($p = .056$). Given the approach to significance and associated low observed power, this finding may warrant consideration of potential for type II error, with the possibility of significance if the sample were larger. Thus, further examination of this hypothesis is worthwhile to provide a clearer picture concerning the role of effective management of chronic pain in treating veterans with PTSD.

Hypothesis IV predicted significant differences in reduction of depression symptoms between the three cohorts: 1) Veterans with a chronic pain condition and pharmacological

treatment for pain, 2) Veterans with a chronic pain condition without pharmacological treatment for pain, and 3) Veterans with no chronic pain condition and no pharmacological treatment for pain, with Cohort 2 demonstrating significantly less depressive symptom reduction than Cohorts 1 and 3. The MANOVA result supported this hypothesis, showing the significant interaction of time by group on BDI-II scores. This finding indicated significant group differences in depressive symptom reduction across the three cohorts. However, the examination of post-hoc analyses revealed that, while veterans with a chronic pain condition and no pharmacological treatment for pain and veterans with no chronic pain condition and no pharmacological treatment for pain differed most substantially on BDI-II scores across time, these differences were not statistically significant. The results again appear to approach the identified significance level ($p < .05$) with significance observed at .064, warranting consideration that post-hoc analyses were underpowered to detect specific differences over time due to cohort sizes. There is potential for a larger sample size to yield increased power, with a greater ability to detect statistically significant differences.

In considering the significance of differential change in PTSD and depression symptoms between the three cohorts, results appear to be inconclusive, demonstrating some indication for further research. Overall, the results appear support the notion that chronic pain is something that warrants attention in a PTSD treatment setting.

Limitations

Threats to internal validity. Caution should be utilized in interpreting the study results due to threats to internal validity inherent in the study. First, as the present study was a retrospective data analysis, its primary limitation is the inability to utilize random assignment in assigning participants to treatment groups; a requirement for a true experimental design. Thus,

the threat to internal validity exists and causal relationships between chronic pain status and treatment outcomes following CPT cannot be inferred. Second, using self-report and repeated measures of the PCL and BDI-II, may carry various reporting biases due to social desirability, variance in response styles, and memory effects. Third, the small sample size is a limitation of the study. Although the sample size met a-prior power analysis criteria, low statistical power was observed in the interaction effect of the MANOVA analysis. Larger cohort sizes (resulting in larger overall sample size) would have ultimately strengthened the study's ability to detect treatment differences between the three cohorts. Finally, the study may have left influential variables unaccounted for. In using archival data, the selection of variables was restricted. As such, there are likely other, variables that have the potential to influence CPT treatment outcomes for the three cohorts of veterans. Additional forms of chronic pain management, that are unidentifiable via chart review, appear to be the most prominent examples of these potential uncontrolled variables. This may include any non-VA pain medications, pain medications taken without a prescription, use of illicit substances to manage pain, and non-VA complimentary or alternative pain management interventions.

Threats to external validity. The sample reflects a small portion of the veteran population nationwide, which makes generalizability of the findings limited. Although the database originated from various locations nationwide spanning over 20 VA Medical Centers and Community Based Outpatient Centers, certain geographic regions appear largely underrepresented, and as such, generalizability of results is limited. Additionally, to enhance internal validity, various exclusion criteria, including number of CPT sessions completed, time-span of treatment regimen, and initial versus repeated engagement in CPT, were included in

participant selection. Implementation of these criteria certainly provided limitations in generalizability of the results.

Implications for Clinical Practice

There appear to be three primary clinical implications from the present study. First, a strong correlation was observed between PTSD and depression variables in the study. Although the MANOVA controlled for unique contributions of variance in these variables, the strong correlation indicates a significant relationship between PTSD and depression. While the significance of this relationship is certainly not a novel finding, it further supports the existing literature and warrants continued clinical attention towards assessment of depressive symptoms, particularly those with significant associated function impairment (e.g. fatigue/loss of energy) or mortality consequences (e.g. suicidal ideation), in PTSD treatment settings.

Second, the results of significant change in both PTSD and depression symptom across CPT, in all cohorts in the sample, provide support for the utility of CPT in veterans with both PTSD and chronic pain. Although effectiveness cannot be specifically addressed in the current study, results provide preliminary evidence that warrants consideration of effectiveness of CPT in this population.

Lastly, results of the study appear to warrant attention to the comorbid presentation of chronic pain and PTSD, in a PTSD treatment setting. With results initially revealing significant group differences in depression symptom reduction and near significance observed in group differences in PTSD symptom reduction with low observed power, there appear to be preliminary indications of a significant relationship between chronic pain status and CPT treatment outcomes. These indications may warrant attentiveness to the assessment of pain in PTSD treatment setting as well as consideration of concurrent pain management interventions for

veterans with both PTSD and chronic pain. Additionally, these indications may include consideration or exploration of conjunctive pain treatment interventions as well as specific attention towards or assessment of pain severity.

Future Research Directions

There are several potential future avenues for research, stemming from the results of the present study. Most significantly, examination of the efficacy of CPT in conjunction with a chronic pain intervention, in reducing both PTSD and depressive symptoms in veterans with PTSD and chronic pain, appears warranted. Similarly, a consideration that untreated chronic pain has the potential to interfere with or limit the effectiveness of CPT, resulting in less symptom reduction, appears warranted. Examination of efficacy would require use random assignment of participants to chronic pain treatment groups as well as inclusion of a control group. These changes in methods would allow for causal interpretation of the effect of chronic pain status on treatment outcomes following CPT.

Additionally, future research may wish to explore effects of various types of pain management, in conjunction with CPT or another identified PTSD treatment, in veterans with both PTSD and chronic pain. This may include use of acupuncture, biofeedback for pain, physical therapy, implementation of anti-inflammatory diets and more. Given the VA initiative to reduce prescriptions of opioid medications, exploration of alternative pain management interventions appears particularly warranted.

Finally, incorporation of metrics indicating pain severity could be useful in future research. This may include pain metrics utilized in primary care or other medical settings as well as pain-specific questionnaires. In any format, capturing severity of pain in conjunction with

PTSD treatment intervention could provide rich data regarding the implications of pain on PTSD treatment.

Conclusion

The results from the present study support existing literature on the efficacy of CPT in the reduction of PTSD symptoms for veterans, regardless of their chronic pain status. Similarly, the present study provides support for previous research findings regarding the relationship between PTSD and depression symptoms, while also demonstrating significant symptom reduction following CPT, across all cohorts in the study. The inconclusive findings regarding the differential degree of symptom reduction among veterans with PTSD and chronic pain with or without pain treatment appear to warrant consideration of pain in PTSD treatment settings as well as further exploration of the topic in future research. These future research endeavors may continue to explore a variety of pain management interventions in conjunction with PTSD treatments, utilizing an experimental design with a large sample, in the hope that a comprehensive treatment regimen or best practice is identified for veterans who hold both chronic pain and PTSD diagnoses.

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Appendix A

PTSD CheckList – Civilian Version (PCL-C)

Client's Name: _____

Instruction to patient: Below is a list of problems and complaints that veterans sometimes have in response to stressful life experiences. Please read each one carefully, put an "X" in the box to indicate how much you have been bothered by that problem *in the last month*.

No.	Response	Not at all (1)	A little bit (2)	Moderately (3)	Quite a bit (4)	Extremely (5)
1.	Repeated, disturbing *memories, thoughts, or images* of a stressful experience from the past?					
2.	Repeated, disturbing *dreams* of a stressful experience from the past?					
3.	Suddenly *acting* or *feeling* as if a stressful experience *were happening* again (as if you were reliving it)?					
4.	Feeling *very upset* when *something reminded* you of a stressful experience from the past?					
5.	Having *physical reactions* (e.g., heart pounding, trouble breathing, or sweating) when *something reminded* you of a stressful experience from the past?					
6.	Avoid *thinking about* or *talking about* a stressful experience from the past or avoid *having feelings* related to it?					
7.	Avoid *activities* or *situations* because they *remind you* of a stressful experience from the past?					
8.	Trouble *remembering important parts* of a stressful experience from the past?					
9.	Loss of *interest in things that you used to enjoy?*					
10.	Feeling *distant* or *cut* off from other people?					
11.	Feeling *emotionally numb* or being unable to have loving feelings for those close to you?					
12.	Feeling as if your *future* will somehow be *cut short*?					
13.	Trouble *falling* or *staying asleep*?					
14.	Feeling *irritable* or having *angry outbursts*?					
15.	Having *difficulty concentrating*?					
16.	Being *"super alert"* or watchful on guard?					
17.	Feeling *jumpy* or easily startled?					

PCL-M for DSM-IV (11/1/94) Weathers, Litz, Huska, & Keane National Center for PTSD - Behavioral Science Division

Appendix B

Beck Depression Inventory--Second Edition

Veteran's Name: _____ Date: _____

Veteran's DOB: _____ Last four digits of SSN: _____

Staff: _____ Location: _____

This questionnaire consists of 21 groups of statements. Please read each group of statements carefully, and then pick out the one statement in each group that best describes the way you have been feeling during the PAST TWO WEEKS, INCLUDING TODAY. Select the number beside the statement you have picked. If several statements in the group seem to apply equally well, select the highest number for that group.

1. SADNESS
- ☐ 0. I do not feel sad.
- ☐ 1. I feel sad much of the time.
- ☐ 2. I am sad all the time.
- ☐ 3. I am so sad or unhappy that I can't stand it.

2. PESSIMISM
- ☐ 0. I am not discouraged about my future.
- ☐ 1. I feel more discouraged about my future than I used to be.
- ☐ 2. I do not expect things to work out for me.
- ☐ 3. I feel my future is hopeless and will only get worse.

3. PAST FAILURE
- ☐ 0. I do not feel like a failure.
- ☐ 1. I have failed more than I should have.
- ☐ 2. as I look back, I see a lot of failures.
- ☐ 3. I feel I am a total failure as a person.

4. LOSS OF PLEASURE
- ☐ 0. I get as much pleasure as I ever did from the things I enjoy.
- ☐ 1. I don't enjoy things as much as I used to.
- ☐ 2. I get very little pleasure from the things I used to enjoy.
- ☐ 3. I can't get any pleasure from the things I used to enjoy.

5. GUILTY FEELINGS
- ☐ 0. I don't feel particularly guilty.
- ☐ 1. I feel guilty over many things I have done or should have done.
- ☐ 2. I feel quite guilty most of the time.
- ☐ 3. I feel guilty all of the time.

6. PUNISHMENT FEELINGS
- ☐ 0. I don't feel I am being punished.
- ☐ 1. I feel I may be punished.
- ☐ 2. I expect to be punished.
- ☐ 3. I feel I am being punished.

7. SELF-DISLIKE
- ☐ 0. I feel the same about myself as ever.
- ☐ 1. I have lost confidence in myself.
- ☐ 2. I am disappointed in myself.
- ☐ 3. I dislike myself.

8. SELF-CRITICALNESS
- ☐ 0. I don't criticize or blame myself more than usual.
- ☐ 1. I am more critical of myself than I used to be.
- ☐ 2. I criticize myself for all my faults.
- ☐ 3. I blame myself for everything bad that happens.

9. SUICIDAL THOUGHTS OR WISHES
- ☐ 0. I don't have any thoughts of killing myself.
- ☐ 1. I have thoughts of killing myself, but I would not carry them out.
- ☐ 2. I would like to kill myself.
- ☐ 3. I would kill myself if I had the chance.

10. CRYING
- ☐ 0. I don't cry anymore than I used to.
- ☐ 1. I cry more than I used to.
- ☐ 2. I cry over every little thing.
- ☐ 3. I feel like crying, but I can't.

11. AGITATION
- ☐ 0. I am no more restless or wound up than usual.
- ☐ 1. I feel more restless or wound up than usual.
- ☐ 2. I am so restless or agitated that it sometimes hard to stay still.
- ☐ 3. I am so restless or agitated that I have to keep moving or doing something.

12. LOSS OF INTEREST
- ☐ 0. I have not lost interest in other people or activities.
- ☐ 1. I am less interested in other people or things than before.
- ☐ 2. I have lost most of my interest in other people or things.
- ☐ 3. It's hard to get interested in anything.

13. INDECISIVENESS
- ☐ 0. I make decisions about as well as ever.
- ☐ 1. I find it more difficult to make decisions than usual.
- ☐ 2. I have much greater difficulty in making decisions than I used to.
- ☐ 3. I have trouble making any decisions.

14. WORTHLESSNESS
- ☐ 0. I do not feel I am worthless.
- ☐ 1. I don't consider myself as worthwhile and useful as I used to.
- ☐ 2. I feel more worthless as compared to other people.
- ☐ 3. I feel utterly worthless.

15. LOSS OF ENERGY
- ☐ 0. I have as much energy as ever.
- ☐ 1. I have less energy than I used to have.
- ☐ 2. I don't have enough energy to do very much.
- ☐ 3. I don't have enough energy to do anything.

16. CHANGES IN SLEEPING PATTERN
- ☐ 0. I have not experienced any change in my sleeping pattern.
- ☐ 1. I sleep somewhat more than usual.
- ☐ 2. I sleep somewhat less than usual.
- ☐ 3. I sleep a lot more than usual.
- ☐ 4. I sleep a lot less than usual.
- ☐ 5. I sleep most of the day.
- ☐ 6. I wake up 1-2 hours early and can't get back to sleep.

17. IRRITABILITY
- ☐ 0. I am no more irritable than usual.
- ☐ 1. I am more irritable than usual.
- ☐ 2. I am much more irritable than usual.
- ☐ 3. I am irritable all the time.

18. CHANGES IN APPETITE
- ☐ 0. I have not experienced any change in my appetite.
- ☐ 1. my appetite is somewhat less than usual.
- ☐ 2. my appetite is somewhat greater than usual.
- ☐ 3. my appetite is much less than before.
- ☐ 4. my appetite is much greater than usual.
- ☐ 5. I have no appetite at all.
- ☐ 6. I crave food all the time.

19. CONCENTRATION DIFFICULTY
- ☐ 0. I can concentrate as well as ever.
- ☐ 1. I can't concentrate as well as usual.
- ☐ 2. It's hard to keep my mind on anything for very long.
- ☐ 3. I find I can't concentrate on anything.

20. TIREDNESS OR FATIGUE
- ☐ 0. I am no more tired or fatigued than usual.
- ☐ 1. I get more tired or fatigued more easily than usual.
- ☐ 2. I am too tired or fatigued to do a lot of the things I used to do.
- ☐ 3. I am too tired or fatigued to do most of the things I used to do.

21. LOSS OF INTEREST IN SEX
- ☐ 0. I have not noticed any recent changes in my interest in sex.
- ☐ 1. I am less interested in sex than I used to be.
- ☐ 2. I am much less interested in sex now
- ☐ 3. I have lost interest in sex completely.